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VASCULAR DISORDERS OF THE LIMBS.¹

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THE subject for review tonight is "Vascular Disorders of the Limbs". There is not enough time at our disposal to discuss these conditions at length. Indeed, it will only be possible to cover the ground by giving a rather sketchy outline of the principal disorders of this kind, and I propose to deal, for practical reasons, with the clinical aspects and the treatment of these disorders mainly.

You are aware that in 1936 Sir Thomas Lewis published the result of many years' investigations and researches into this subject, in the form of a book entitled "Vascular Disorders of the Limbs".⁽¹⁾

¹ Read at a meeting of the New South Wales Branch of the British Medical Association on June 24, 1937.

This is a standard work, and full of valuable information.

Lewis's approach is mainly physiological.

There have also been a great number of reports published throughout the world on the subject, and I propose to refer shortly to some of the results of treatment reported on, and to mention some of my experiences in treatment.

I do not propose to deal at length with the circulation and its control.

You will recall that the rate of blood flow in the limbs is mainly regulated by the smaller arteries and arterioles. The capillaries and minute venules, and the large arteries and the veins are less active in this process. There are in the limbs, also, direct connexions between the arterioles and venules, through arteriolo-venous anastomoses; the walls of these anastomosing vessels are very muscular and richly innervated, and when open, allow a rapid flow of blood from arterioles to venules. The small arteries and arterioles and anastomoses maintain the

temperature of the limbs, of the digits, and of the palms of the hands and soles of the feet.

These vascular reactions are controlled by the central nervous system through the sympathetic vasomotor nerves, distributed in the limbs by way of the mixed nerves of the brachial and lumbar plexuses. These sympathetic nerves maintain vaso-constriction. If a sympathetic nerve is cut, the tonic action of the vaso-constrictor sympathetic nerve is lost, and the vessels dilate. There may be vaso-dilator nerves, but little is known of these.

In health, general vaso-constriction of the cutaneous vessels of the body occurs under the influence of emotion or fear, or of painful or very cold stimuli applied to the surface of the body. This sympathetic nervous action is reinforced by the simultaneous releases of hormones (such as adrenaline), into the blood stream. The suprarenal gland secretion is increased in circumstances of general vaso-constriction, and acts with the sympathetic nerves in governing vascular tone.

The above facts represent what occurs in healthy persons, and give no indication as to the reason for pathological states.

The nutritional requirements of the tissues are met by an exchange through the walls of the minute blood vessels. There are fluctuations in the size of the smallest vessels, and the fluctuations are controlled by the products of local metabolism, which act on the vessels.

It is interesting to observe that all the blood vessels of the limbs shrink when they are cooled; this local effect is important in the minor maladies, such as chilblains, Raynaud's phenomenon, frostbite *et cetera*, but is certainly not the cause of local arterial degeneration or thrombo-angiitis.

Method of Examining the Vessels of the Limbs.

Many methods are employed for examining the vessels of the limbs. Actually, there is little need for elaborate investigations. A great deal can be learnt by using the eyes and the hands. I shall leave out all description of the more elaborate tests.

Skin Temperature.—The digits provide the most sensitive temperature gauge of the circulation to the skin of the limbs. In order to ascertain whether the circulation is defective in one limb (when there is doubt on the point), the limbs may be tested by simple palpation, after being exposed under identical conditions for ten minutes; or both limbs may be immersed in warm water, about blood heat, for a few minutes, then thoroughly dried, and symmetrically disposed for observation. The limb in which the circulation is more active will remain warm longer. As a rule, the cooler limb has the smaller cutaneous blood supply; owing to vascular defect in the arteries of the limb. These vessels may be diseased, or their tone may be increased. Occasionally, the warm limb is the affected one, as when it has lost its sympathetic nerve supply.

Skin Colour.—The colour of the skin mainly depends on the state of the circulation of the skin itself, the minute vessels of the skin, and the plexuses of the minute venules in the subpapillary region. It frequently is an important guide to the state of the flow of blood in the limb as a whole.

Tests of Vessels.—Examination of the vessels of the limbs should be made with the limbs equally and thoroughly warm. The limb vessels should be followed upwards. If pulsation ceases in the line of an artery, the significance is obvious. Pachon's oscillogram may be applied at similar levels of the two limbs, and the extent of the oscillations of the aneroid manometer compared.

The patency of the radial and ulnar (or posterior tibial and *dorsalis pedis*) arteries can be determined by obliterating one of the main vessels, say the radial, while the hand is held aloft. The hand is then lowered to see if it quickly fills with blood; if it does not do so, obviously the radial artery constitutes almost the entire source of supply; and the hand will flush red on the release of pressure on the radial artery. When the blood flow to foot or hand is very poor, the extremity becomes unusually pale when lifted above the level of the body.

Spasm.—Immersing the part in warm water will relax spasm of the arteries. Spinal and local anaesthesia also block the sensory and sympathetic nerves, with resulting vaso-dilatation. If there is a spasmodic obstruction to blood flow it may be suddenly relaxed in this way. When an artery is blocked by structural disease, warming the part causes its temperature to rise slowly, and not so high as it normally would.

A simple method of ascertaining the extent of arterial disease is to warm the limb by fully immersing it in water at 35° C. (95° F.) for ten minutes; raise the limb above the body level and massage it till pale. Now apply a pressure, through a sphygmomanometer cuff, higher than systolic pressure. Maintain this arrest of circulation for five to ten minutes while the limb remains in the warm bath. The limb is then lifted from the bath, dried, and its circulation released. In normal limbs the skin becomes brightly coloured to the ends of the digits in two to five seconds. In diseases of the vessels this reaction is slower, taking up to one minute.

Note the part of the limb unflushed at the end of five seconds; this will indicate the territory affected by arterial disease. When disease is present, the flushing of the limb lasts much longer than in the normal area, where it lasts only a few minutes at most.

Tests of Vasomotor Nerves.—Warm the trunk or another limb. General vasomotor tone is lost when the temperature is raised. If the vasomotor nerves to one arm are paralysed, and the subject is in a cool room, the fingers on that side will be warmer at first, but as vaso-dilatation occurs, it will involve the normal limb only, and the fingers on the normal side will become hotter. Vasomotor tone may be abolished from the skin by heating the body, or by soaking one arm or both feet in stirred water at 44° C. (110° F.). By these means the blood is warmed, returns to the heart, and thence passes to the heat-regulating centres.

Disorders of the Limb Circulation.

As there has been a certain amount of confusion in the differentiation of some of these vascular disorders of the limbs, I shall briefly mention some of the less

serious conditions in order to indicate the necessity for their exclusion when a diagnosis is sought. In serious disorders, such as arteriosclerosis and *thrombo-angiitis obliterans*, some of the signs mentioned in connexion with the less serious disorders may occur.

Disorders of the limb circulation are: chilblains, acrocyanosis or acroasphyxia, erythrocyanosis, erythralgia, intermittent claudication, *thrombo-angiitis obliterans* and diabetic arteritis. Time permits of my barely mentioning some few points about each condition.

Chilblains.

Chilblains are described by Lewis as a form of chronic inflammation, modified by occurring in a skin almost habitually cold. The inflammation in urticaria is due to the release of a natural histamine-like substance from damaged cells into the tissue spaces. The three stages of urticaria, a central red spot or line due to a local dilatation of minute vessels, the wheal, and the bright flare in the surrounding skin, can be reproduced by directly introducing histamine into the skin.

The sudden onset of chilblains suggests some similar mechanism. Chilblains result from some personal idiosyncrasy. The rate of clotting of the blood was said by Wright to be prolonged in subjects of chilblains. This was the reason for giving calcium in treatment, and I am told that intravenous injection of calcium gluconate or lœnulate is effective; acetylcholine also has the reputation of relieving chilblains.

Acrocyanosis or Acroasphyxia.

Acrocyanosis or acroasphyxia occurs mainly in young women. The hands, usually to a little above the wrists, are persistently of higher colour than normal. They are usually cyanosed when cold, bright red when very cold or warm, often mixed red and purple. The hands are often colder than normal and the palms sweaty. The malady often occurs in persons of the lethargic type. The minute vessels of the skin are very dilated; the pulses in the main arteries are normal. The constriction is in the small arteries or arterioles of the skin.

Lewis states that the increased tone of the vessels is due to a fault in the vessels themselves, for it is not quickly relieved by anæsthetizing the nerves, as it would be if it were vasomotor. He holds that it is not a vasomotor disorder, as is often thought, but a disorder in which the arterioles of the skin are unusually susceptible to cold. Professor Lambie will shortly publish details of a case which illustrates this condition very fully.

Treatment.—Treatment consists in avoiding exposure and in maintaining the warmth of the hands.

Erythrocyanosis of the Leg.

Erythrocyanosis of the leg may probably be regarded as one example of a chilblain phenomenon; it is often regarded as a vasomotor phenomenon. It is almost entirely confined to young women. A deep vascular discoloration of the skin occurs, mainly of cyanotic tints in advanced grades; a single large area is usually involved, some inches long by a few inches broad. The disorder generally

affects the legs and ankles. The skin is tender, and is sensitive to friction and warmth. The minute vessels are dilated, the arterioles constricted. The condition is not due to a vasomotor disorder, it is inflammatory; histological examination reveals perivascular lymphocytic infiltrations of the skin and subcutaneous tissues. The malady came in with short skirts; it is increased in winter and decreases in summer. It has been fairly common in England during the craze for shorts and no stockings.

Erythralgia.

Erythralgia is Lewis's term for a redness of the skin accompanied by a peculiar form of tenderness. Pain is induced in this condition by stretching the skin between the fingers or by friction or warming. Cooling relieves the pain, unless a low temperature is reached, when pain is again produced. It is a burning pain. The condition is one of inflammation; it can be readily produced by burning the skin. Sunburn causes similar sensations, and has a similar pathology. Erythralgia occurs in many conditions: in stages of cellulitis, frost-bite and chronic chilblains of the foot; in erythrocyanosis; and in gross arterial disease of the leg, when gangrene threatens.

Erythralgia is not a vasomotor disorder; it always results from a local process. The cause is the release from the cells into the skin of an unknown substance (not histamine), which lowers the threshold of the pain nerve endings to various forms of stimulation. The treatment of erythralgia is that of the underlying condition.

Erythromelalgia.⁽¹⁾

Erythromelalgia is the term invented by Weir Mitchell to describe a condition of burning pain and redness in the dependent leg. The condition is merely a symptom of many disorders, and the name is best discarded for "erythralgia". Both erythromelalgia and erythralgia appear to be symptoms of some local disorder.

Loss of Sympathetic Innervation; Vasomotor Palsy.

It may be in order now to mention loss of sympathetic innervation. Division of the sympathetic nerves leads to vaso-dilatation, redness and increased warmth in the skin area supplied, also to cessation of sweating and to pilomotor paralysis. The affected area of skin is not always warmer than the skin on the normal side; that is, if the normal side is warmed or the whole body is warmed, the normal limb may become warmer than the innervated limb, but the denervated limb maintains a more constant temperature, since there are no fluctuations in vasomotor tone.

I mention vasomotor palsy here because most disturbances in the circulation of the limbs have at one time or another been attributed to that cause; quite wrongly, it appears.

Trophic Changes.—Lewis states that the majority of so-called trophic changes in the fingers, toes and limbs in chronic nervous disorders are really the result of disuse. Atrophy, loss of sensibility, decline in the blood flow and fall of temperature are the factors causing "trophic changes", and the ulcers, slow-healing and so on, which are noted in chronic

nervous disorders. The "trophic changes" in poliomyelitis, progressive muscular atrophy, syringomyelia and hemiplegia are simply due to local mechanical effects, and are not vasomotor disorders. This is an interesting statement, from one who has studied the conditions much.

Arteriosclerosis (Endarteritis Obliterans) and Diabetic Arteritis.

I come now to speak of the more serious vascular disorders of the limbs. There is a great similarity in the symptoms of the different diseases of the arteries of the limbs.

Arterial disease of the elderly and diabetic is bilateral, but the symptoms are usually unilateral, for months or years. Early symptoms may be a sense of coldness, heaviness, slight numbness or tingling, cold feet at night, or cramps. The disease is advanced by the time the symptoms appear. Numbness of the toes and discoloration or swelling of the limbs may occur, but pain is the most frequent and insistent symptom. The pain may be in the toes, in the feet or in the whole leg, or in the fingers or hands in *thrombo-angiitis obliterans*.

Intermittent claudication, due to the diminished blood supply, is felt in the calf, in the front of the leg, or in the foot. The pain may be continuous, or may occur only on movement. Discoloration of the foot or leg, associated with coldness of the part, occurs. The skin is often sensitive to friction and to warmth, and the patient may keep his feet outside the bed clothes on account of their being hot. Either warmth or friction or hanging the leg down may induce burning pain. The increased tension of the skin causes pain, which may be very intense. The pain causes disuse, and defective nutrition leads to weakness and atrophy.

The pulses of the foot, and often of the leg, may not be found. Capillary pulsation will not occur on heating the toes, and natural vaso-dilatation will be diminished or absent. In arteriosclerosis and in diabetes X ray examination may reveal calcareous deposits outlining the vessels of the foot or leg. Indolent ulceration of the nail bed of the great toe, or ulceration of a corn on the toe or side of the foot, may occur. The lesion is often traumatic, due to an ingrowing toe nail, or the use of a caustic for a corn. In other patients necrosis begins in a blister or simple abrasion. The lesion progresses, the cyanotic tint of the toes increases from purple to dark brown, and is accompanied by a loss of sensation in the skin. The tissues blacken, an odour of decomposition is observed, and the necrotic area becomes sharply marked off from the remainder of the toe. The necrotic tissue may die and shrink, and may separate with the enclosed bone after some months; or it may become moist and ooze. Infection, ascending lymphangitis, or cellulitis of the leg may occur, and this leads to septicæmia.

Thrombo-angiitis Obliterans.

Thrombo-angiitis obliterans, which was described by Buerger⁽³⁾ in 1908, occurs in young men mostly. It was said to be more common in Russian or Polish Jews. In the arteries the media is uncalcified. The intima shows no atheroma. Within the main elastic lamina lie great masses of organized tissue, permeated

by small vascular channels. There may be fresh blood clot. The walls of the affected arteries show signs of chronic inflammation in the outer coats, which are often bound firmly to surrounding veins, nerves and other structures. The lesion involves the main artery and all its chief branches throughout the leg. Both legs are affected, and the arms to a less extent; rarely the visceral arteries are affected. The process is gradual, and numerous arterial anastomoses occur throughout the limb. The veins show isolated or migrating thrombi, and are themselves obliterated.

Thrombo-angiitis is believed to attack the vessel wall, causing inflammation and secondary thrombosis. Buerger thought it was due to cigarette smoking, and many have supported his view of the aetiology. The youngest patient I have seen was only twenty-six years of age; he was otherwise a healthy young man. Both legs were affected, the popliteal artery being occluded on one side. The condition also occurs in women, rarely.

Symptoms of Thrombo-angiitis.—The symptoms of thrombo-angiitis are similar to those of arteriosclerosis. The popliteal and femoral pulses may be obliterated in both legs; the pulses of the arms may be affected, and Raynaud's phenomenon may occur in the fingers, because of disease of the arteries of the hand. All four limbs may be seriously affected, and all four limbs may require amputation. Mental symptoms occur in some patients; whether these are due to pain or to cerebral vascular lesions is uncertain. A history of painful red lumps in the legs may be obtained, these being due to migrating phlebitis in the veins.

Buerger drew attention to a sign called erythromelia, a flushing of skin of the foot when the leg hangs down. This flushing is replaced by definite blanching of the skin when the foot is elevated at varying angles between 90° and 180°; the greater the angle before the blanching appears, the better is the degree of circulatory efficiency.

Diagnosis.—The history and complete physical examination of the patient, X ray photographs of the legs, the application of the Wassermann test and of blood sugar tests, if necessary, should decide the diagnosis in most cases. It is often impossible to differentiate between arteriosclerosis (or *endarteritis obliterans*) and *thrombo-angiitis obliterans*; even *post mortem* the diagnosis is difficult, and Professor Inglis has told me that clear-cut cases of one or the other of these two diseases are rare, most pathological specimens being referred for examination when the disease has reached a very advanced stage. It is said that arteriosclerosis occurs at a later age than thrombo-angiitis, and that calcification never occurs in thrombo-angiitis. A study of the records of Sydney Hospital reveals this difficulty in diagnosis. Clinically, the diagnosis has to be made largely on the age of onset, but in the present state of our knowledge the treatment is much the same in either case.

Treatment of Arterial Disorders of the Limbs.

Prophylaxis.

The same prophylaxis applies to most disorders of the limb circulation. It is important to keep the body and the feet and hands warm, as far as possible.

On no account should hot bottles or heat be applied to the feet. The best way to warm the feet is to warm the body and thighs. If the general health is affected, it should be attended to.

Diabetes.—Diabetics require treatment for their condition. I do not know whether insulin treatment is said to diminish the tendency towards arteritis; presumably it should. I do not prescribe insulin for diabetics as a rule, if the urine can be kept sugar-free, or nearly so, on an adequate diet. One is aware of the risks of a raised blood sugar content, but when there is no glycosuria insulin is rarely prescribed continuously, nor can one guarantee freedom from complications by giving insulin. In a recent case I did not find that a course of insulin, of two months' duration, had any effect in permanently diminishing the blood sugar content in a patient with a high blood sugar content.

In one patient, H.V., a male, aged forty-seven years, the figure for the blood sugar concentration was 114 when the patient was fasting; it was 205 one hour after the patient had taken glucose, and 176 two hours after he had taken glucose. Ten units of insulin were given three times a day before meals for four weeks, and five minims three times a day for four weeks. One week later the blood sugar concentration figure was 0.128 when the patient was fasting, 0.238 one hour after he had taken glucose, and 174 two hours after he had taken glucose. Incidentally, this patient had been having large doses of insulin for some eight weeks prior to the above treatment. Possibly the more prolonged action of protamine insulin may be helpful in such cases.

Tobacco.—Smoking should be stopped by all patients suffering from vascular disorders of the extremities, though I still feel doubtful about the relationship between tobacco and chronic arterial disease. Pharmacologically, nicotine is said to cause vaso-constriction. I cannot see why cigarettes alone should cause trouble.

Care of the Feet.—You are all familiar with the necessity for extreme care in nail cutting and corn cutting. Lewis says that people suffering from arterial disorders should be as careful of their limbs as if they were made of porcelain. Unfortunately, the loss of sensibility in the part makes injury more likely than it is in the normal limb. Obviously, the patient should not do anything which may aggravate the disease. When claudication occurs, the patient should not walk far enough at one stretch to cause pain. Of course, patients do so in spite of advice to the contrary, just to see if the pain is still there.

Local Treatment.

If the skin develops discoloration or tenderness, the foot should be kept at rest, at body level, under close observation. Desiccation may be encouraged by a current of warm air driven by a fan. Dry sterile gauze, arranged so as not to adhere to the injured skin, should be applied. For infection in the early stages, Dakin's solution may be applied. Pain may be relieved by keeping the foot up and keeping it cool. Morphine may be necessary.

As soon as symptoms of vascular disease arise, a spell of four to six weeks in bed may be helpful in

warding off the more serious consequences for a time. While the patient is in bed, various treatments may be tried, such as Buerger's exercises, 0.1 gramme of acetyl choline twice a day, hot douching or electrical warming of the whole limb. Lewis states that acetyl choline has no effect, because it is destroyed on being injected subcutaneously. Martindale, in the "Extra Pharmacopoeia", states that it has a powerful vasodilator effect, and personal experience with patients suffering from Raynaud's syndrome and chilblains supports this view.

In 1935, Brown and Allen,⁽⁴⁾ from the Mayo Clinic, claimed that in 2,000 patients suffering from occlusive vascular disease, the best results were obtained by giving intravenous injections of typhoid vaccine. They described the treatment of a number of patients by alternate pressure and suction. The affected limb was enclosed in a box, and a negative pressure of 60 to 80 millimetres was alternated with a positive pressure of 20 millimetres; four cycles of pressure were given in each minute. Briefly, they did not consider the results very satisfactory, equally good results being obtained by other methods, such as intravenous injections of typhoid vaccine and intravenous injections of hypertonic saline. They held the view that rest in bed was the most important single measure in cases where serious symptoms occurred.

Barker and others, in 1935, reported the results of treatment with various tissue extracts: firstly, pancreatic tissue extract; secondly, extract of skeletal muscle (myoston); thirdly, muscle adenosin phosphoric acid; and fourthly, adenosin phosphoric acid. All caused a prolongation of the claudication time when injected intramuscularly; that is, a prolongation of the time before the onset of claudication, following a given exercise. The pancreatic tissue extract and extract of skeletal muscle were more effective than the other preparations.

These extracts were injected intramuscularly once a week, for eight to sixteen doses, causing an improvement in intermittent claudication greater than that caused by any other method. They did not relieve the pain of ulcer or gangrene due to vascular occlusion. There was no evidence that they caused vascular dilatation, and it was suggested that they supplied some substance which the muscle lacked. This is particularly interesting, since Lewis⁽⁵⁾ had shown, in 1931, that the pain in intermittent claudication was due to stimulation of the pain nerve endings by the action of some chemical substance released from the muscle fibres, in proportion to their contraction; when the circulation was deficient, this substance accumulated during exercise to the level sufficient to cause pain.

Elliott and Evans⁽⁶⁾ have shown that this substance is in all probability lactic acid. They further found that the ingestion of 100 grammes of glucose, or the injection of 20 units of insulin, increased exercise tolerance in the ischaemic limb, which observation may be of use therapeutically. Personal experience indicates that the following may be of use in treatment: 283.5 grammes (ten ounces) of 5% hypertonic saline solution, given intravenously twice a week for five weeks or more; 0.1 gramme of acetyl choline, given hypodermically twice a day for some

weeks; muscle extract, given once a week or more. If the condition does not respond to rest and general treatment, surgical treatment may be necessary. Periarterial sympathectomy has fallen under the ban of Lewis, and I believe is generally regarded as ineffectual. However, Hamilton Bailey⁽⁷⁾ reported excellent results from this procedure in a case of Raynaud's disease.

The last two or three lumbar sympathetic ganglia (second to fourth), with the intervening stretches of sympathetic cord, may be removed. A preliminary test of spinal anaesthesia or "Novocain" injection may be made to ascertain how much vaso-dilatation may be expected. Lumbar sympathectomy appears to have given quite good results in some cases.

Treatment of Thrombo-angiitis.

I shall mention a few cases to illustrate the variability in the course of these complaints, and the remarkable improvement sometimes obtained by prolonged conservative treatment. As in all disorders of the extremities, a sane conservatism is necessary in treatment.

A patient, C.H.B., was diagnosed as suffering from *endarteritis obliterans* and thrombo-angiitis. In July, 1928, he complained of a bad toe; his doctor removed the nail and made two incisions into the great toe of the right foot on July 9. A few days later the toe turned septic. The patient was admitted to hospital on August 9. A raw septic area was present at the site of the toe nail of the right big toe, the terminal phalanx was exposed, cellulitis was present in all the toes, and the patient complained of severe pain. This condition continued until May, 1929, in spite of rest and local treatment. On May 7, 1929, right lumbar ramisection was performed by Mr. George Bell. After operation the colour returned to the toes of the right foot much more quickly than before, but not so quickly as on the left side. The ulceration cleared up, but all the toes of the right foot remained dark red in colour.

From 1929 to 1933 much pain was felt in the legs, feet and toes. In March, 1933, there was oedema and redness of the lower third of both tibiae and oedema of the left ankle. In December, 1933, there was pain and swelling of the left great toe. No pulsation was felt in the *dorsalis pedis*, but the left posterior tibial artery pulsed. The left foot became cold when the patient walked. Injections of acetyl choline were given daily for three weeks, causing slight improvement.

In January, 1934, "Padutin" was injected thrice weekly without any improvement resulting. In April, 1934, an ulcer was noted on the lateral aspect of the left great toe. Six intravenous injections of typhoid vaccine were given, commencing with 5,000,000 units. In June ulceration had extended, but the pain was less. In July, 1934, the lower part of the left sciatic nerve was injected in the upper part of the popliteal space with a 2% solution of "Novocain". The pain in the toes was relieved and marked flushing of the left foot was observed when it was in the dependent position; on elevation the foot became pale. There was a temporary decrease in ulceration and pain, but both recurred.

In August the left femoral artery was stripped for approximately one and a half inches (periarterial sympathectomy). Again there was temporary improvement. In September and October ten intravenous injections of ten ounces each of 5% saline solution were given three times a week. In June, 1935, there was a small area of ulceration only. Eight more intravenous injections of saline solution were given. In November a sequestrum was removed. In February, 1936, the ulceration of the toe was soundly healed. In April, 1937, there was some complaint of cramp in the feet, but no return of ulceration. The patient is now able to report.

A patient, S.W.A., was seen on August 22, 1930, complaining of sudden acute pain in the left leg from the middle of the thigh to the ankle, and numbness of the left foot. The lower third of the left leg and foot were very cold and blue. Pulsation could not be felt in the arteries of either foot. There was a collapsing radial pulse. The reaction of the blood to the

Wassermann test was positive, the result being "+++". Pulsation could not be felt in the left femoral artery, and only slightly in the right femoral artery. Amputation through the left thigh was performed. The vessels were small but patent; little bleeding occurred. The pathological report stated that the arteries were not calcareous nor markedly hardened. The lumina were visible at the proximal ends, but were pin-point in size farther down. Microscopically the proximal portion showed little change. The posterior tibial artery showed fibroblastic proliferation of the intima and adhesions of different parts of the walls, so that several lumina replaced a single lumen. A diagnosis of *thrombo-angiitis obliterans* was made.

In October, 1930, a severe attack of pain occurred in the right foot and leg. There was loss of sensation below the middle third of the right leg. A black slough occurred at the edge of the nail bed of the second toe. The great toe was colder than the other toes. In five days the whole of the great toe and second toe were gangrenous. Next day amputation was performed through the right thigh just above the knee; the only vessels requiring ligation were the femoral and the *arteria comitans nervi ischiadici*; the latter artery appeared to have a comparatively large lumen.

The pathological report was as follows: Thickening was present in the intima of the arteries; the muscularis showed fenestrations occupied by strands of fibrous tissue which seemed to run radially; another vessel showed eccentric intimal thickening and the same changes in the muscularis; still another showed almost entire obliteration of the lumen, and close to one side a narrow, slit-like path. The condition was very suggestive of Buerger's disease.

Arteriotomy.—The removal of a portion of a diseased artery for relief of pain has been recently advocated anew by Leriche in an article published in *Surgery, Gynecology and Obstetrics*, of February, 1937. There is not time to deal with the indications for this treatment.

Amputation.—It is not possible to make set rules for guidance, but usually amputation is necessary when gangrene is extensive, when it involves several toes or one or more toes and the adjacent part of the foot, or when massive gangrene of the foot occurs. When a toe has become gangrenous, the gangrene having appeared to settle down to a given line, if further spread of the gangrene is definitely observed, amputation is usually desirable. Quick extension often means infection, and, if it involves deep tissues, causing swelling, or marking out lymphatics, the limb requires immediate amputation.

In the elderly and -diabetic, when conservative treatment has failed, amputation through the lower third of the thigh is indicated in order to obtain a sound artery; but in thrombo-angiitis amputation at lower levels may be safe.

If, in reactive hyperaemia, a bright colour appears in the warmed heel within fifteen seconds of circulatory release, amputation may be just above the ankle; if the colour takes this time, or little longer, to reach the bases of the affected toes, these alone should be amputated.

Vascular Spasm; Ergot Poisoning.

Vascular spasm due to ergot poisoning is of interest, since it shows how readily vascular degeneration may be produced by poisons. Now a rare disorder, it exemplifies spasmodic obstruction of vessels, which may lead to necrosis in the distal tissues of the limb. In this condition, after the patient has been consuming infected rye for some days or weeks, tingling pain or painful cramps occur, followed by a feeling of coldness and numbness of the skin. Bruises or blisters or infection beneath the nails occurs, and some days

or weeks later darkening of the skin, indicating gangrene, occurs, usually symmetrically, in the hands or feet. It is of interest that the injection of 19 millilitres of ergotamine tartrate within a week was followed by gangrene of both feet in a man, and that four injections of 0.25 milligramme caused gangrene and death in a middle-aged woman. The drug should not be used in patients with vascular disease. The pharmacopoeial dose of ergotamine tartrate is 0.5 milligramme. These effects of the injection of ergot must be borne in mind in trying to elucidate the aetiology of arterial disease of the limbs. They show how quickly permanent effect may be induced by vascular poisons.

There are two other causes of vascular spasm. The first is increased arterial tone due to local cold. Exposure of a single limb to cold by immersion in water at a temperature of 15° C. or 20° C. causes a decrease in the size of the main arteries and pulses, and in the size of the veins, owing to the direct action of cold on the vessels. The second cause of vascular spasm is local arterial spasm due to impact. Even large arteries may exhibit a state of extreme spasm after an impact, such as a bullet wound, and this spasm may last for some hours. Arterial spasm is often invoked to explain symptoms, but the condition of the artery is often one of structural disease, and not purely spasmodic.

Actually, vascular spasm, or local contraction, has not been demonstrated, except from the causes alluded to, and as a result of Raynaud's disease. Neither is there any evidence that structural disease and vascular spasm occur together, as Lewis⁽¹⁾ points out. However, ophthalmoplegic migraine is surely a case in point, and one can think of no other adequate explanation for some transient hemiplegias and some varieties of *angina pectoris*.

Raynaud's Phenomenon.

Transient discoloration of the fingers and symmetrical gangrene occur in a variety of maladies. In all these conditions there are serious local faults, and it is doubtful whether Raynaud's syndrome is ever due purely to over-action of the vasomotor nerves. Disease of the digital arteries occurs in *thrombo-angiitis obliterans* and in diffuse scleroderma. The simplest form of Raynaud's phenomenon begins in childhood. Single attacks occur at rare intervals. The subjects are usually thin and have as a rule suffered from cold extremities or chilblains. One or more fingers may be affected by the arrest of circulation, usually the second to the fifth digits; sometimes the thumb, or the whole hand, or the lower part of the forearm is affected. Similar conditions may occur in the feet; they rarely occur in the nose, ears or cheeks. Exposure to cold water or cold air usually brings on the attacks. One finger, or the tips of the fingers, may be affected. Excitement, fear or anger may bring on an attack when the hands are cold. The attack starts at the tip of the finger, the arteries there closing, and as it spreads upwards the finger pales, becomes greyer, and may be blanched and waxy in appearance; sometimes the fingers are cyanotic. In either case circulation is arrested. In half an hour the finger becomes numb, and may

grow painful during prolonged attacks or on very cold days. Recovery is indicated by a redness invading the finger from its base; and tingling, followed by warmth, occurs quickly afterwards.

The condition is common in its minor forms in New South Wales. Unless it is disabling or progressive, symptomatic treatment and avoidance of cold may be helpful. The patient should keep warm by every means in his power, wear warm clothes and gloves; he should rest in bed if necessary for a few days. Acetyl choline and muscle extracts may be used. Severe symptoms may necessitate periarterial sympathectomy or cervical sympathectomy. The response to these measures is frequently dramatic. In our climate, however, these operations can rarely be necessary for any except very severe attacks.

Diseases with Local Nutritional Changes.

Diseases with local nutritional changes occur in women mainly. Attacks similar to those of Raynaud's disease occur, and increase in frequency and duration. Partial recovery may occur. The hands seem cyanotic, with patches of bright redness; but the red patches are not warm, and they turn blue again. This is called intermittent leakage, the spasm being relaxed temporarily. Atrophy of the soft tissues and bone occurs; sclerodactyly may develop. Areas of necrosis appear at the tips of the fingers; scales or plugs of dead skin, which separate slowly and painfully, or necrotic areas occur at the borders of the nail. Injuries heal slowly. Dusky cold chilblains may occur. The terminal phalanges may become shortened. Gangrene of a large part of the finger is very rare. When warm, fingers so affected show reduction in the volume of arterial pulsation, and capillary pulsations cannot be elicited. The condition is chronic. Diffuse scleroderma develops in many patients.

The digital arteries are narrowed with thickening of the intimal coat. The condition is not a vasomotor disorder, and is not cured by the destruction of the sympathetic blood supply to the limb.

Treatment.

The treatment of these diseases is the same as the treatment for Raynaud's phenomenon. The patient should avoid exposure out of doors in wet, windy or cold weather, and also cold bathing; he should wear warm clothes. The temperature of the room should be kept at 21° C. (70° F.), and the hands should be kept under the bed clothes at night. Gloves, thick socks *et cetera* and gum boots should be worn. The hands, when cold, should be warmed by warm water, and they should be freely used. Massage should be ordered, and a liberal diet should be given. Vasodilator drugs have little effect. Thyroid extract is sometimes useful.

Sympathectomy.—Sympathectomy improves the circulation in limbs so affected, but its effects diminish in time. Lewis speaks very highly of it, but records a few disadvantages. The inferior cervical ganglion should be removed, the object being to denervate the hand. The inferior cervical ganglion comprises the ganglia corresponding to the seventh and eighth cervical and the first dorsal segments fused together.

Other varieties of Raynaud's phenomenon are: Raynaud's phenomenon due to local injury or vibration, Raynaud's phenomenon due to thromboangiitis, Raynaud's phenomenon due to acute thrombosis, Raynaud's phenomenon due to a cervical rib. There are also rare conditions of unknown aetiology, such as bilateral gangrene in the young and elderly, and bilateral gangrene with haemoglobinuria from cold. Cold causes a haemolysin in the blood stream to unite with the blood cells; haemolysis occurs, and haemoglobin is passed in the urine. There is not time to discuss these conditions at length.

Conclusion.

The points to be noted especially are the widespread effect of cold, of trauma and of poisons such as ergot and nicotine. These three factors may eventually prove to be important in the aetiology of vascular disorders of the limbs, and may lead to the discovery of other aetiological factors.

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PLACENTAL INFECTION IN INDUCED LABOUR, WITH SPECIAL REFERENCE TO ITS RELATION- SHIP TO FETAL AND NEO-NATAL MORTALITY.

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In an earlier paper⁽¹⁾ we described the frequent infection of the placenta with *Bacillus coli* following the induction of labour with the rectal tube. Moreover, such infection of the placenta was associated with fetal or neo-natal death. The material studied was derived from twenty-five cases of eclampsia, and the suggestion was made that the very high infantile mortality following the surgical induction of labour in eclampsia was partly due to the infection of the placenta and the fetus by the operation of induction. The many reports in the literature of *Bacillus coli* infections of the new-born and the frequency with which, in the cases we studied, infection of the placenta by coliform organisms was associated with stillbirth or neo-natal death led us to believe that such infections of the new-born frequently arose from the contaminated placenta while the fetus was still in utero.

The writings on the subject of placental infection and infection of the fetus *in utero* are not extensive and do not indicate that the surgical induction of labour may be important in such happenings.

Harris and Brown⁽²⁾ (1927) studied the infection of the uterine contents in fifty cases of Caesarean section. The material for the cultures was obtained by swabbing the lower uterine segment through the uterine incision. In all those cases in which more than a few hours had elapsed since the onset of labour, bacteria were present. Premature rupture of the membranes and vaginal examinations were the factors mentioned as increasing the likelihood of bacterial invasion of the uterus. No information was given as to the infection of the placenta or of the child.

Kobak⁽³⁾ (1930) made foetal blood cultures from blood withdrawn from the cord during the third stage of labour in 377 births. In 37 cases the blood cultures were positive; in five, two types of bacteria were cultivated from the blood. Only aerobic methods of cultivation were employed; the bacteria most often isolated were diphtheroids, *Bacillus coli*, *Staphylococcus albus*, streptococci, and *Bacillus faecalis alkaligenes*. These positive foetal blood cultures were associated with early rupture of the membranes. Kobak considered that infection of the placenta resulted from bacterial contamination of the liquor amnii; in three instances he recorded the cultivation of bacteria from the liquor. In one case *Bacillus coli* and streptococci, identical with those from the cord blood, were obtained from the liquor amnii. In three instances in which the child failed to survive, similar organisms to those cultivated from the cord blood were obtained from the fetus.

In 1915 Slemons⁽⁴⁾ drew attention to what he termed "placental bacteriemia" as a cause of infantile death. Three out of twenty-seven dead infants examined by him were associated with placentas infected with streptococci (type not stated). In one case sections of the placenta revealed, in addition to the streptococci, bacteria resembling *Bacillus welchii*. In these three cases labour had been prolonged. The placental infection was without serious effect on the mother. In most of the recent literature, however, there is no reference to this type of placental infection as the cause of infantile deaths. Cruickshank⁽⁵⁾ (1930), in his discussion of the causes of neo-natal death, made no mention of it, and Bourne and Williams⁽⁶⁾ (1932), under the heading "placental causes" of foetal death, did not discuss infection apart from syphilis. Fleming⁽⁷⁾ (1933), who laid especial stress on infection as a cause of infantile mortality, did not discuss this point.

The possibility of an infected liquor amnii resulting in infection of the child while still in utero has received more attention. Brown⁽⁸⁾ (1921) observed the association of pneumonia in the new-born with premature rupture of the membranes, and cited cases in which infants who died within a few hours of birth showed an advanced degree of pneumonia. Furthermore, these lung infections

were frequently due to coliform organisms. Fleming (1933) also drew attention to such possibilities.

The Scope of the Present Investigation.

In this paper are recorded the results of the bacteriological examination of 114 placentas, 46 of which were derived from cases in which labour had been induced with the rectal tube; the remainder were from cases in which surgical induction of labour had not been practised. In addition, in those cases in which the child failed to survive, cultures were also obtained from the foetus in most instances.

The technique employed for the examination of the placenta was as follows. The placenta was delivered in as careful a manner as possible and placed immediately in a sterile metal container with a firmly fitting flat lid and taken forthwith to the laboratory. The lid was removed from the container and used as a tray to hold the placenta. Portion of the fetal surface of the placenta was seared and blood was withdrawn with a Pasteur pipette from two or more of the larger vessels and inoculated in approximately one cubic centimetre quantities into 30 cubic centimetres of brain broth (Kracke and Teasley⁽⁹⁾) and into 15 cubic centimetres of anaerobic tryptic broth.⁽¹⁰⁾ The placenta was then turned over and two or more separate portions of the maternal surface were well seared. Portions of tissue directly beneath the seared areas were removed and added to media similar to those receiving the blood. The making of the cultures was completed within thirty minutes of the delivery of the placenta.

When the child was stillborn or died within ten days of birth, similar cultures to those made from the placenta were made from its heart's blood, spleen, lung and occasionally from the liver. Unfortunately, except in one instance, it was not possible to make these cultures from the infant until ten hours after death, and in one or two cases twenty-four hours had elapsed. The possible influence of this delay on the results obtained is discussed later.

The Frequency and Nature of Placental Infection Following the Tubal Induction of Labour.

Placental infection was detected 2.5 times more often following the surgical induction of labour than in those cases in which no such operation had been performed. More striking still was the difference in the nature of the infections in the two groups (see Table I). Of the 46 placentas from cases in which induction had been practised, 28 were infected, 13 with *Bacillus coli* and 4 with *Bacillus welchii*, indicating the faecal origin of the infections. Two of the 13 placentas infected with *Bacillus coli* were also infected with *Bacillus welchii*. Among the 68 controls, however, only 16 yielded growth, and in no instance were the organisms of a characteristic faecal type.

Another important point of difference between the two groups was the different frequency with which the infecting organisms were cultivated from the blood of the fetal vessels of the placenta. Among the control group infection of the placental vessels was never detected, but among the 46 induced cases organisms were cultivated from the blood of these vessels in 14 instances. There were two other striking differences between the two groups: the absence of mixed infections of the placenta among the non-induced controls and the rarity of aerobic infections in this same group. Of the 16 strains of bacteria isolated from the placentas from normal labours only three were able to grow in the presence of air. Table I gives the various types of bacteria isolated from the two groups; brief descriptions of the strains are given in Appendix II.

We do not consider that the ten strains of *Staphylococcus albus* and the six strains of aerobic diphtheroids isolated from the placentas of the patients whose labours were induced were contaminants introduced during the making of the cultures, since such bacteria were not obtained from any of the control group. We believe these bacteria were enabled to grow up into the uterus following the operation of induction. They were possibly faecal in origin or more likely came originally from

TABLE I.
The Frequency and Nature of Placental Infection.

Group.	Total Number Infected.	Number Infected with More than One Type of Bacterium.	Number Showing Growth from Blood of Fetal Vessels of Placenta.	Number Infected with											
				Bacillus coli.		Bacillus welchii.		Staphylococcus albus.		Aerobic Streptococci.		Anaerobic Streptococci.		Aerobic Diphtheroids.	
				A.	M.	A.	M.	A.	M.	A.	M.	A.	M.	A.	M.
A. 46 placentas from cases in which induction was performed.	28 (61%)	16 (35%)	14 (30%)	2	11	2	2	1	9	1	9	4	5	0	6
B. 68 placentas from cases in which induction was not performed.	16 (24%)	0 (0%)	0 (0%)	0	0	0	0	0	0	8	0	4	0	0	8

A. = Occurring alone.

M. = Occurring as an element in a mixed infection.

The figures in parentheses give the percentage frequencies.

the skin and were enabled to invade the placenta together with the faecal organisms introduced, since in all but two instances they were present in the placenta in association with typical varieties of faecal bacteria.

Factors Predisposing to Infection of the Placenta Following the Tubal Induction of Labour.

Although the above results leave no doubt that induction of labour with the rectal tube often results in infection of the placenta, it does not occur in every instance. The important factor appears to be the length of time elapsing between the insertion of the rectal tube and the delivery of the child. If this interval, which we have termed the post-induction interval, is prolonged, there is an increased risk of infection of the placenta. The average post-induction interval for the 28 cases showing infection of the placenta was 63 hours, whereas for the 18 cases in which the placenta was sterile the post-induction interval was 31 hours. Table II shows a similar correlation when the cases are grouped according to whether they were first pregnancies or not, or according to the fate of the child. In 12 of the 46 cases the post-induction

believe, to the length of time elapsing between the operation of induction and the emptying of the uterus, rather than to such factors as lowered resistance to infection conditioned by the maternal toxæmia.

The stage of the pregnancy at which labour was induced also appeared to be without great influence on the infection of the placenta. Among the nine cases in which the duration of the pregnancy was 32 weeks or less, there were six showing infection of the placenta; among the other 37 cases in which pregnancy was further advanced, 22 showed infection.

The Correlation between Placental Infection and the Death of the Child.

Among the 46 cases studied there were six in which the child was born so prematurely that its chance of survival was extremely remote. These six cases, therefore, have been excluded from the present analysis. In Tables III and IV are included only those cases (40 in number) in which the weight of the child at birth was three and a half pounds or over and its length seventeen inches or over.

TABLE II.
The Relationship of the Length of the Post-Induction Interval to the Infection of the Placenta.

Group.	Number in Group.	Number Infected.	Average Post-Induction Interval in Cases with Infected Placentas.	Number Sterile.	Average Post-Induction Interval in Cases with Sterile Placentas.
Total number of placentas examined	46	28	63 hours.	18	31 hours.
Placentas from primiparae	29	19	69 hours.	10	40 hours.
Placentas from multiparae	17	9	52 hours.	8	19 hours.
Placentas from cases in which the child survived	31	17	57 hours.	14	36 hours.
Placentas from cases in which the child failed to survive	15	11	73 hours.	4	11 hours.

interval was 18 hours or less, and in no instance had infection of the placenta occurred, whereas among the 34 cases in which the post-induction interval was over 18 hours, there were 28 which showed infection of the placenta.

The condition of the mother, *per se*, did not appear to influence the frequency of infection of the placenta to any significant degree. Among 21 cases in which the indication for the induction was albuminuria, there were ten in which the placenta was sterile; for these the average post-induction interval was 28 hours, while for the 11 cases in which the placenta was infected it was 60 hours.

Although the other groups of cases (classified according to the maternal condition) are too small for statistical analysis, they suggest that the length of the post-induction interval is the important factor, rather than the condition of the mother. Thus among eight cases of post-maturity there were five non-infected cases with an average post-induction interval of 21 hours, as against three infected cases with an average post-induction interval of 86 hours. Among seven cases of pre-eclampsia or eclampsia, there was only one with a post-induction interval less than 34 hours, and each placenta of this group was infected owing, we

From a consideration of Table III it is difficult to escape the conclusion that infection of the placenta following the tubal induction of labour was associated with an increased risk to the life of the child. Mixed infection of the placenta was a greater danger to the child than infection by a single organism. The presence of organisms in the blood of the large foetal vessels of the placenta was associated with a strikingly high infantile death rate, especially if more than one type was present.

TABLE III.
The Relationship between the Bacteriological State of the Placenta and Fetal and Neo-natal Mortality in the Case of 40 Infants which were not Markedly Premature at Birth.

Bacterial State of Placenta.	Number of Cases.	Number of Cases in which Child Failed to Survive.
Sterile	15	1
Infected	25	7
Infected with only one type of organism	11	2
Infected with more than one type of organism	14	5
Large foetal vessels infected	11	7
Large foetal vessels infected with only one type of organism	7	3
Large foetal vessels infected with more than one type of organism	4	4

Not all the organisms isolated from these placentas appeared to be a danger to the child. In Table IV are set out the relationship between the various types or combination of types of bacteria infecting the placenta and foetal or neo-natal death.

TABLE IV.

The Relationship between the Types of Organisms Infecting the Placenta and Foetal and Neo-natal Mortality.

Bacterial State of Placenta.	Number of Cases.	Number of Cases in which Child Failed to Survive.
Infected with <i>Bacillus coli</i> and/or aerobic non-hemolytic streptococci	13	7
Foetal vessels infected with <i>Bacillus coli</i> and/or aerobic non-hemolytic streptococci	8	6
Infected with organisms other than <i>Bacillus coli</i> and/or aerobic non-hemolytic streptococci	11	1
Infected with <i>Bacillus coli</i> either alone or in mixed culture	10	5
Foetal vessels infected with <i>Bacillus coli</i> either alone or in mixed culture	6	4
Infected with aerobic non-hemolytic streptococci either alone or in mixed culture	8	5
Foetal vessels infected with aerobic non-hemolytic streptococci either alone or in mixed culture	5	5
Infected with anaerobic streptococci either alone or in mixed culture	9	0

Although the numbers in Table IV are too small to enable one to draw definite conclusions from them, they suggest that infection of the placenta with *Bacillus coli* and non-hemolytic aerobic streptococci was a source of danger to the life of the child. When these organisms were present in the blood of the large foetal vessels of the placenta, the association between such infection and death of the child either shortly before or after birth was very much greater. In the two cases in this series in which the child survived in spite of the presence of *Bacillus coli* in the blood of the large foetal vessels of the placenta, the organisms were present in pure culture, and a considerable time elapsed before growth was visible in the cultures. In one instance three days' incubation and in the other four days' incubation was necessary before the cultures were cloudy; in all the other cases in which infection with *Bacillus coli* was present, the cultures, whether from placental tissue or blood, showed evidence of growth after twenty-four hours' incubation.

An attempt was made to obtain evidence that the death of the child might be due to infection by the same organisms as were found in the placenta. Owing to hospital regulations it was not possible to obtain material from the dead child until ten or more hours after death. This delay appears to have been unimportant from the point of view of allowing *post mortem* invasion in the case of the neo-natal deaths, since in every case in which *Bacillus coli* was isolated from more than one situation the strains from any one case were identical according to biochemical reactions and agglutinin absorption tests.

Before the infant's body was opened with sterilized instruments, the skin was cleansed with 1% lysol, followed by alcohol and ether. The heart

and portions of lung, spleen and sometimes liver were removed and placed in sterile Petri dishes. The material so collected was transferred immediately to the bacteriological laboratory and the necessary cultures were put up. If the collection of any particular tissue was unsatisfactory, so that contamination was likely to occur, that material was not employed, which accounts for the blanks (-) that appear in Table V.

In 15 out of the 46 cases studied the child failed to survive; from 12 of these infants material was obtained *post mortem*. Table V gives the particulars relative to infection of the placenta and of the foetus. If we consider first the seven cases in which the foetus was dead at birth, we find that in two (Cases IV and V) the placenta and foetus were both sterile, in two (Cases XXIV and XLI) the same types of bacteria were isolated from both the placenta and the foetus. In Case XXII the placenta was infected, both tissue and blood of large foetal vessels, but the cultures from the foetus remained sterile. In this case the foetus was macerated and it would appear possible, if not probable, that the foetal heart ceased to beat before the infection of the placenta was sufficient to cause infection of the foetal circulation. In Case VIII four types of organisms were obtained from both the placental tissue and blood, but from the spleen of the child only two of these types (*Bacillus coli* and diphtheroids) were recovered, and from the lung *Bacillus coli* together with *Bacillus welchii*. In Case XVIII, in which the placental cultures yielded only *Bacillus coli*, *Bacillus welchii* was found in addition to the coliform organism in cultures made from the heart's blood, the spleen and the lung of the child. The presence in the cultures from the foetus in these two cases of an organism additional to those isolated from the placenta was due, we believe, to premature respiration and consequent aspiration into the lungs of contaminated liquor amnii (see following section).

Among the five cases in which the child died shortly after birth, the types of organisms cultivated from the foetal tissues were in every instance also obtained from the placenta. In two cases organisms which were recovered from the placenta were not obtained from the foetus.

The above results suggest that the bacteria detected in the foetus were derived from the placenta or from the same source as that from which the placenta was infected, and that the presence of bacteria, especially *Bacillus coli* and aerobic streptococci, in the blood of the large foetal vessels of the placenta usually indicated a similar infection of the foetus, which in some cases at least was probably responsible for its death. It might be argued that maternal disease was responsible for the infantile deaths. An examination of the case notes does not support this, however. In a number of cases the induction was performed on account of the presence of symptoms of toxæmia, but in most cases the toxæmia was mild; only one case of eclampsia occurred in the series.

TABLE V.

The Bacteria Cultivated from the Placenta and from the Child in Twelve Cases in which the Child Failed to Survive.

Case Number.	Post-induction Interval in Hours.	Fate of Child.	Bacteria from Placental Tissue.	Bacteria from Fetal Vessels of Placenta.	Bacteria from Child, Post Mortem.			
					Heart's Blood.	Spleen.	Lung.	Liver.
IV	11	Macerated, premature.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.
V	10	Stillborn.	Nil.	Nil.	Nil.	Nil.	Nil.	Nil.
VIII	27	Stillborn.	<i>Bacillus coli</i> , non-hemolytic streptococci, <i>Staphylococcus albus</i> , diphtheroid.	<i>Bacillus coli</i> , non-hemolytic streptococci, <i>Staphylococcus albus</i> , diphtheroid.	—	<i>Bacillus coli</i> , diphtheroid.	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	—
XI	40	Died two hours after birth. Premature.	<i>Bacillus coli</i> , <i>Staphylococcus albus</i> .	<i>Staphylococcus albus</i> .	Nil.	Nil.	Nil.	—
XV	72	Died 19 hours after birth. Premature.	<i>Bacillus coli</i> , <i>Streptococcus viridans</i> , <i>Staphylococcus albus</i> .	<i>Bacillus coli</i> .	—	<i>Bacillus coli</i> , <i>Streptococcus viridans</i> .	<i>Bacillus coli</i> , <i>Streptococcus viridans</i> , <i>Staphylococcus albus</i> .	—
XVIII	63	Stillborn.	<i>Bacillus coli</i> .	<i>Bacillus coli</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	—
XXI	150	Died 11 hours after birth.	<i>Bacillus coli</i> , <i>Streptococcus viridans</i> , <i>Bacillus weickii</i> .	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .	—	<i>Bacillus coli</i> , <i>Streptococcus viridans</i> , <i>Bacillus weickii</i> .	—
XXII	96	Macerated.	<i>Bacillus coli</i> , diphtheroid, <i>Staphylococcus albus</i> .	<i>Bacillus coli</i> , diphtheroid.	Nil.	Nil.	Nil.	—
XXIV	144	Macerated.	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .	<i>Streptococcus viridans</i> .
XXVI	44	Died 24 hours after birth.	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .	<i>Bacillus coli</i> , <i>Bacillus weickii</i> .
XXXVII	42	Died five days after birth.	<i>Bacillus coli</i> , non-hemolytic streptococci.	<i>Bacillus coli</i> , non-hemolytic streptococci.	<i>Bacillus coli</i> .	<i>Bacillus coli</i> .	<i>Bacillus coli</i> .	Nil.
XLI	75	Stillborn, slightly macerated.	<i>Streptococcus hemolyticus</i> (not Group A), <i>Streptococcus viridans</i> .	<i>Streptococcus hemolyticus</i> (not Group A), <i>Streptococcus viridans</i> .	<i>Streptococcus hemolyticus</i> (not Group A), <i>Streptococcus viridans</i> .	<i>Streptococcus hemolyticus</i> (not Group A), <i>Streptococcus viridans</i> .	Nil.	Nil.

Case XXXVII was of especial interest.

The child in this instance, apart from its small size, appeared normal at birth (its weight was four pounds four ounces and its length was nineteen inches), but the cultures from the placental tissue and from the blood of the large fetal vessels of the placenta yielded *Bacillus coli* and non-hemolytic streptococci. On the fourth day of life the child was jaundiced and died on the following day. Immediately after death a culture was made from the heart blood, and sixteen hours later cultures were made from the spleen, liver and lung. Except that from the liver, which remained sterile, all the three *post mortem* cultures yielded pure cultures of *Bacillus coli*.

In this case at least it appears that death was due to an infection acquired while the foetus was still

in utero. In this instance the induction had been carried out on account of pyelitis coupled with albuminuria, but the degree of toxæmia exhibited by the mother was mild and it was not considered to have had a detrimental effect on the child.

Ravina⁽¹¹⁾ (1935) has reported a somewhat similar infection to this. He described a baby who was deeply jaundiced at birth and who died five days later. This infant was shown to be suffering from a generalized infection due to *Bacillus coli*, but in this case labour was short and there had been no interference.

At the beginning of this paper mention was made of Kobak's finding in 377 cases in which blood cultures were made from blood withdrawn from the cord during the third stage of labour. In 37 of his cases the blood cultures were positive, but in this series all but four of the babies survived. It is not possible to compare these results with those obtained by us, as unfortunately Kobak's cases were not grouped according to the amount of interference in the conduct of the labour. Furthermore, he does not state the amount of blood used for each blood culture; if a large quantity of blood was used he might have detected the presence of fewer organisms in the blood than was possible with the technique we employed.

Infection of the Liquor Amnii.

The presence in the lungs of some of the dead children of organisms not found in the cultures from the blood of the foetal vessels of the placenta (see Table V, Cases VIII, XVIII and XXI) led us to believe that the *liquor amnii* might be contaminated with organisms not present in the placenta and that in these cases the aspiration of contaminated *liquor amnii* might be partially responsible for the infection of the child.

In three cases a sample of *liquor amnii* was obtained at the time the rectal tube was inserted; in each instance the fluid was sterile. In two instances we were able to obtain specimens of the *liquor amnii* some time after the operation of induction. In each case the *liquor amnii* was infected and, furthermore, organisms were cultivated from it that could not be detected in the foetal vessels of the placenta.

In Case XVI Caesarean section was performed ninety-six hours after the induction. A specimen of *liquor amnii* was obtained for culture immediately the uterus was opened. The cultures from it yielded *Bacillus coli* and an anaerobic streptococcus. In this case *Bacillus coli* alone was cultivated from the placenta.

In the second case (XXI) a specimen of *liquor amnii* was obtained under aseptic conditions when the membranes were ruptured, 130 hours after the induction. From this specimen were cultivated *Bacillus coli*, *Bacillus welchii* and *Streptococcus viridans*. The same three organisms were obtained twenty-four hours later from the placental tissue; from the blood of the foetal placental vessels *Streptococcus viridans* alone was cultivated. The child died eleven hours after birth. Cultures from the heart blood yielded *Streptococcus viridans* in pure culture (corresponding to the blood culture made at birth from the placental vessels), but the cultures from the lung yielded the three types of organisms that had been cultivated from the *liquor amnii*.

The Identity of the Strains from the Placenta and Liquor Amnii with those from the Fœtus.

If, as we believe, the infection of the placenta (and probably also the *liquor amnii*) following the tubal induction of labour often leads to infection of the fœtus, then the strains of any given type of organism cultivated from the placenta and corresponding fœtus should be identical. With the coliform organisms isolated this was so in every instance. The biochemical tests carried out are detailed in Appendix II, where are given also the

results of agglutination and agglutinin-absorption tests. In every instance the strains of *Bacillus coli* isolated from the placenta, the liquor and the fœtus were identical culturally, biochemically and serologically. Among ten strains of *Bacillus coli* isolated from ten different placentas which were compared, there were no two identical. For the organisms other than *Bacillus coli* we were content to record their cultural similarity and in a few instances their biochemical reactions. In every case the cultures from the placenta and the corresponding fœtus appeared similar.

Relationship of Placental Infection to Maternal Morbidity.

The tendency to discount placental infections and to treat them as of the nature of contaminations is not supported when we analyse our material to see what relationship, if any, existed between infection of the placenta and illness in the mother. Of the 46 placentas from cases in which induction of labour had been practised, 22 were from women who showed in the puerperium a febrile reaction of 37.8° C. (100° F.) or more. In 19 of these 22 infection of the placental tissue was present. The samples of tissue, as we have before stated, were taken from the maternal side of the placenta after its surface was seared. In the case of the non-febrile mothers, nine, only out of the 24 placentas were proved by the bacteriological technique that we employed to be infected. Further, the organisms found in the placental tissues in the febrile cases were often definitely pathogenic organisms; whereas those found exclusively in the placental tissue in the non-febrile cases were anaerobic cocci, Gram-positive cocci, diphtheroids and *Staphylococcus albus*, organisms not accepted as being usually pathogenic. When we turn to the organisms found in the foetal vessels of the placentas, it is found that the frequency of infection is not so very different in the febrile and non-febrile mothers. The foetal vessels of the placentas of nine out of twenty-two febrile women were found to be infected, while the foetal placental vessels of five non-febrile women out of twenty-four were likewise found to be infected. That is to say, there is a very high correlation between fever in the mother and infection of the placental tissues, but a much less significant correlation between the febrile reaction in the mother and infection in the foetal placental vessels. This is to be expected, for infection of the foetal vessels of the placenta is not of necessity directly associated with infection of the mother, since these placental vessels are portion of the foetal circulatory system.

A study of some of the individual cases yields further evidence that the organisms infecting the placenta and *liquor amnii* following the tubal induction of labour may give rise to infection of the mother.

In Case XVI Caesarean section was carried out ninety-six hours after the induction. *Bacillus coli* was isolated from both the placental tissue and the blood of the foetal placental vessels; from the *liquor amnii* collected at opera-

tion *Bacillus coli* and an anaerobic streptococcus were obtained. The operation wound became infected; the organisms responsible for this infection were *Bacillus coli* and an anaerobic streptococcus of exactly similar type to those obtained at operation from the placenta and liquor amnii.

In Case XLII one of the organisms cultivated from the placental tissue was a non-hæmolytic streptococcus. Two days after delivery, when the mother's temperature was 38.9° C. (102° F.), a streptococcus similar to that obtained from the placenta was cultivated from her blood.

In these two cases it is difficult to escape the conclusion that the febrile illness of the mother was due to spread of infection from the placenta, which had been infected by the operation of induction.

Summary and Conclusions.

1. The tubal induction of labour is frequently associated with infection of the placenta.

2. The type of such infection differs from that found associated with normal labour. In the normal labours studied the placentas have not shown *Bacillus coli*, aerobic non-hæmolytic streptococci or *Bacillus welchii*; moreover, they have shown no mixed infections. In the placentas derived from tubally induced labours *Bacillus coli*, aerobic non-hæmolytic streptococci and *Bacillus welchii* have been common. Mixed infections have also been frequent.

3. If there be a prolonged interval between the induction of labour and the delivery of the child, the risk of infection of the placenta is greatly increased. Placental infection is not dependent to any great extent on the maternal condition or on the age of the fetus.

4. The infections of the placenta with *Bacillus coli*, aerobic non-hæmolytic streptococci and *Bacillus welchii* have been associated with a high infantile mortality. Mixed infections of the placental tissue and infections of the large fetal vessels of the placenta are especially serious for the infant.

5. The bacteria cultivated from the dead infants are usually similar to those obtained from the placenta. When additional organisms are present in the fetus, their source is probably an infected liquor amnii. When *Bacillus coli* has been found in the placenta and in the body of the dead infant, the strains isolated have been biochemically and serologically identical, suggesting a single disease process.

6. There is a high correlation between fever in the mother and infection of the placental tissue.

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for bacteriological examination should be immediately available. The splendid cooperation of the nursing staff also greatly assisted us.

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APPENDIX I.

Brief Notes of the Forty-Six Cases in which Labour was Induced with the Rectal Tube.

CASE I.—R.G. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred ninety hours after induction. From the tissue of the placenta an anaerobic streptococcus, a diphtheroid and a Gram-negative filamentous organism were grown; cultures from the blood of placental vessels remained sterile. The child (five pounds six ounces, twenty inches) survived. The mother's temperature rose to 37.9° C. (100.2° F.) the day following induction, and to 37.2° C. (99° F.) the day following delivery.

CASE II.—M.T. was pregnant with her seventh child. The indication for induction was albuminuria. Delivery occurred fifty-two hours after induction. From the tissue of the placenta *Staphylococcus albus*, an anaerobic streptococcus and *Bacillus coli* were cultivated; cultures from the blood of placental vessels remained sterile. The child (5 pounds five and a half ounces, nineteen and a half inches) lived. The mother's temperature rose to 38.2° C. (100.8° F.) the day after delivery and to 37.6° C. (99.8° F.) on the day of delivery (two days after induction).

CASE III.—V.V. was pregnant with her sixth child. The indication for induction was albuminuria. Delivery occurred twelve hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (seven pounds four ounces, twenty-one inches) lived. The mother's temperature remained normal.

CASE IV.—A.P. was pregnant with her first child. The indication for induction was fetal death. Delivery occurred eleven hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The fetus was premature and macerated; cultures from the heart blood, the spleen, the liver and the lung remained sterile. The mother's temperature did not rise above 37.5° C. (99.6° F.).

CASE V.—M.H. was pregnant with her third child. The indication for induction was ante partum eclampsia. Delivery occurred ten hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (eight pounds, twenty-three inches) was stillborn. Cultures from heart blood, spleen and lung remained sterile. The mother's temperature did not rise above 37.2° C. (99° F.).

CASE VI.—I.M. was pregnant with her seventh child. The indication for induction was albuminuria. Delivery occurred twelve and a half hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (three pounds seven ounces, thirteen inches) was born with abdominal ascites and died half an hour after birth; no cultures were obtained post mortem. The mother's temperature did not rise above 37.4° C. (99.3° F.).

CASE VII.—E.R. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred ten hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (one pound six ounces, twelve and a half inches) died two days after birth. No cultures were obtained post mortem. The mother's temperature remained normal.

CASE VIII.—E.G.F. was pregnant with her fourth child. The indications for induction were disseminated sclerosis, chronic nephritis and albuminuria. Delivery occurred twenty-seven hours after induction. *Staphylococcus albus*, a non-hæmolytic streptococcus, *Bacillus coli* and a diphtheroid were isolated from both the tissue of the placenta and the blood of the placental vessels. The child (four pounds twelve ounces, nineteen inches) was stillborn. *Bacillus coli* and a diphtheroid were isolated *post mortem* from the spleen, and *Bacillus coli* and *Bacillus welchii* from the lung of the infant. The patient ran a septic temperature for one month following delivery.

CASE IX.—B.R. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred twenty-four hours after induction. Cultures from the tissue of the placenta yielded *Staphylococcus albus* only; those from the blood of the placental vessels remained sterile. The child (seven pounds three ounces, twenty-one inches) lived. The mother's temperature was normal.

CASE X.—E.D. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred sixty-six hours after induction. *Staphylococcus albus* and *Bacillus coli* were cultivated from the tissue of the placenta, *Bacillus coli* alone from the blood of the placental vessels. The child (four pounds eleven ounces, eighteen inches) lived. The mother's temperature was normal.

CASE XI.—P.C. was pregnant with her second child. The indication for induction was albuminuria. Delivery occurred forty hours after induction. *Staphylococcus albus* and *Bacillus coli* were isolated from the placental tissue, and *Staphylococcus albus* was isolated from the blood of the placental vessels. The child (one pound eight ounces, twelve and a half inches) died two hours after birth. *Bacillus coli* and *Staphylococcus albus* were cultivated *post mortem* from the abdominal fluid; cultures from heart blood, spleen and lung remained sterile. The mother's temperature remained normal.

CASE XII.—D.F.M. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred seventy-six hours after induction. A diphtheroid and a Gram-positive diplococcus were isolated from the tissue of the placenta. Cultures from the blood of the placental vessels remained sterile. The child (five pounds twelve and a half ounces, nineteen inches) lived. The mother's temperature remained normal.

CASE XIII.—R.B. was pregnant with her seventh child. The indication for induction was albuminuria. Delivery occurred eight hours after induction. Cultures from the placental tissue and from the blood of the placental vessels remained sterile. The child (seven pounds fourteen ounces, twenty-one inches) lived. The mother's temperature remained normal.

CASE XIV.—D.C. was pregnant with her first child. The indication for induction was sapremia. Delivery occurred forty-eight hours after induction. An anaerobic streptococcus, *Staphylococcus albus* and a diphtheroid were cultivated from the tissue of the placenta; *Staphylococcus albus* was isolated from the blood of the placental vessels. The child (five pounds nine ounces, twenty-one inches) lived. The mother's temperature was 37.8° C. (100° F.) the day preceding delivery, 38° C. (100.5° F.) four days after, and 37.9° C. (100.3° F.) five days after delivery.

CASE XV.—N.E. was pregnant with her first child. The indication for induction was pyelonephritis. Delivery occurred seventy-two hours after induction. *Bacillus coli*, *Streptococcus viridans* and *Staphylococcus albus* were isolated from the placental tissue; *Bacillus coli* was isolated from the blood of the placental vessels. The child (one pound nine and a half ounces, thirteen inches) died nineteen hours after birth; *post mortem* *Bacillus coli* and *Streptococcus viridans* were grown from the spleen, and *Bacillus coli*, *Staphylococcus albus* and *Streptococcus viridans* from the lung. The mother's temperature was 37.3° C. (99.2° F.) two days before delivery and rose to 38.2° C. (100.8° F.) seven days after delivery.

CASE XVI.—M.R. was pregnant with her first child. The indications for induction were preeclampsia and disproportion. Classical Cesarean section was performed ninety-six hours after induction. *Bacillus coli* was isolated from both the tissue of the placenta and the blood of the placental vessels. *Bacillus coli* and an anaerobic streptococcus were cultivated from the liquor amnii (obtained at operation) and later from the infected operation wound. The child (six pounds six ounces, twenty and a half inches) lived. The mother's temperature was 37.6° C. (99.8° F.) the day before operation (three days after induction), rose to 38.3° C. (101° F.) following operation and to 39.4° C. (103° F.) following the wound infection.

CASE XVII.—E.O.B. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred nineteen hours after induction. An anaerobic streptococcus and a diphtheroid were isolated from the tissue of the placenta. Cultures from the blood of the placental vessels remained sterile. The child (six pounds eleven and a half ounces) lived. The mother's temperature rose to 38.9° C. (102° F.) six days after delivery. There was no other rise in temperature.

CASE XVIII.—L.K. was pregnant with her first child. The indication for induction was ante partum eclampsia. Delivery occurred sixty-three hours after induction. *Bacillus coli* was grown from the tissue of the placenta and also from the blood of the placental vessels. The child (three pounds fifteen ounces, eighteen inches) was stillborn and macerated. *Bacillus coli* and *Bacillus welchii* were isolated *post mortem* from the heart blood, the spleen and the lung. The mother's temperature was 37.8° C. (100° F.) the day the induction was performed, 38.5° C. (101.3° F.) the day of delivery, 39.3° C. (102.8° F.) the day following, and 39.5° C. (103.2° F.) the fourth day after delivery. Blood cultures from the mother were "negative".

CASE XIX.—D.K. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred eighty hours after induction. Cultures from the placental tissue and from the blood of the placental vessels remained sterile. The child (six pounds eight ounces, twenty-two inches) lived. The mother's temperature was normal.

CASE XX.—I.J. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred forty hours after induction. An anaerobic streptococcus was isolated from the tissue of the placenta; the cultures from the blood of the placental vessels remained sterile. The child (five pounds seven ounces, nineteen inches) lived. The mother's temperature rose to 38.3° C. (101° F.) on the twelfth day after delivery.

CASE XXI.—M.N. was pregnant with her first child. The indication for induction was preeclampsia. Delivery occurred one hundred and fifty hours after induction. *Bacillus coli*, *Streptococcus viridans* and *Bacillus welchii* were isolated from the tissue of the placenta; *Streptococcus viridans* only was isolated from the blood of the placental vessels. *Bacillus coli*, *Streptococcus viridans* and *Bacillus welchii* were cultivated from the liquor amnii (obtained by rupture of membranes the day before delivery). The child (three pounds eight and a half ounces, seventeen and a half inches) died eleven hours after birth. *Post mortem*, *Streptococcus viridans* was obtained from the heart blood, and *Bacillus coli*, *Streptococcus viridans* and *Bacillus welchii* from the lung. Cultures from the liver and spleen remained sterile. The mother's temperature remained normal.

CASE XXII.—N.B. was pregnant with her sixth child. The indications for induction were premature rupture of membranes and pyelitis. Delivery occurred ninety-six hours after induction. *Staphylococcus albus*, *Bacillus coli* and a diphtheroid were isolated from the tissue of the placenta; *Bacillus coli* and a diphtheroid from the blood of the placental vessels. The child (three pounds twelve ounces, eighteen and a half inches) was stillborn and macerated. Cultures made *post mortem* from the heart

blood, the spleen and the lung remained sterile. The mother had an irregular temperature for a fortnight following delivery.

CASE XXIII.—B.R. was pregnant with her first child. The indication for induction was preeclampsia. Delivery occurred fifty-five hours after induction. From the tissue of the placenta an anaerobic streptococcus was isolated; cultures from the blood of the placental vessels remained sterile. The child (four pounds six ounces, twenty-one inches) lived. The mother's temperature rose to 37.6° C. (99.8° F.) nine days after delivery, to 38.5° C. (101.4° F.) the eleventh day, to 39.4° C. (103° F.) on the thirteenth day, and was still above 38.8° C. (101° F.) on the twenty-third day. Pelvic cellulitis was present.

CASE XXIV.—N.G. was pregnant with her first child. The indication for induction was breech presentation. Delivery occurred one hundred and forty-four hours after induction. Cultures from both the tissue of the placenta and from the blood of the placental vessels yielded *Streptococcus viridans*. The child (seven pounds fourteen ounces, twenty-one and a half inches) was stillborn and macerated. A streptococcus, biochemically similar to that isolated from the placenta, was obtained at autopsy from the heart blood, the lung, the liver and the spleen of the child. The mother's temperature did not rise above 37.5° C. (99.5° F.).

CASE XXV.—L.R. was pregnant with her first child. The indication for induction was breech presentation. Delivery occurred ninety-six hours after induction. From the tissue of the placenta *Staphylococcus albus* and an aerobic non-hæmolytic streptococcus were isolated. Cultures from the blood of the placental vessels remained sterile. The child (seven pounds eight and a half ounces, twenty-two inches) lived. The mother's temperature rose to 37.9° C. (100.3° F.) on the day of delivery and to 37.8° C. (100° F.) the day after.

CASE XXVI.—E.G. was pregnant with her twelfth child. The indication for induction was preeclampsia. Delivery occurred forty-four hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels yielded *Bacillus coli* and *Bacillus welchii*. The child (two pounds eight ounces, fourteen inches) was premature and died twenty-four hours after birth. *Bacillus coli* and *Bacillus welchii* were isolated at autopsy from the heart blood, the lung, the liver, the spleen and the peritoneal fluid. The mother's temperature rose to 38.5° C. (101.4° F.) the day following induction, and to 37.9° C. (100.2° F.) on the day of delivery.

CASE XXVII.—M.C. was pregnant with her fifth child. The indication for induction was thyrotoxicosis. Delivery occurred forty-seven hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (seven pounds four and a half ounces, twenty inches) lived. The mother's temperature remained normal.

CASE XXVIII.—R.W. was pregnant with her tenth child. The indication for induction was postmaturity. Delivery occurred seventy-two hours after induction. An anaerobic streptococcus and an anaerobic bacillus (not *Bacillus welchii*) were isolated from the tissue of the placenta. Cultures from the blood of the placental vessels remained sterile. The child (seven pounds nine ounces, twenty-one and a half inches) lived. The mother had a swinging temperature for a fortnight following delivery. Blood cultures were "negative".

CASE XXIX.—C.K. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred seventy hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (seven pounds twelve ounces, twenty-one inches) lived. The mother's temperature was 38.4° C. (101.2° F.) the day of delivery and 37.8° C. (100° F.) the second day after delivery.

CASE XXX.—K.P. was pregnant with her first child. The indication for induction was albuminuria. Delivery

occurred twelve hours after induction. Cultures from both the tissue of the placenta and from the blood of the placental vessels remained sterile. The child (seven pounds four ounces, twenty-one inches) lived. The mother's temperature remained normal.

CASE XXXI.—L.C. was pregnant with her second child. The indication for induction was possible postmaturity. Delivery occurred eighteen hours after induction. Cultures from both the tissue of the placenta and the blood of the placental vessels remained sterile. The child (seven pounds eight ounces, twenty-one inches) lived. The mother's temperature remained normal.

CASE XXXII.—J.T. was pregnant with her first child. The indication for induction was postmaturity. Delivery occurred sixteen hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (seven pounds two and a half ounces, nineteen inches) lived. The mother's temperature remained normal.

CASE XXXIII.—C.D. was pregnant with her first child. The indication for induction was postmaturity. Delivery occurred sixteen hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (seven pounds six ounces, nineteen and a half inches) lived. The mother's temperature rose to 39.3° C. (102.8° F.) on the fourth day after delivery and to 39.2° C. (102.6° F.), 39.6° C. (103.4° F.) and 40.2° C. (104.4° F.) on the fifth, sixth and seventh days respectively from some unascertained cause.

CASE XXXIV.—M.F. was pregnant with her sixth child. The indication for induction was albuminuria. Delivery occurred fifty hours after induction. An anaerobic streptococcus was isolated from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (eight pounds fifteen ounces, twenty inches) lived. The mother's temperature did not rise above 37.5° C. (99.6° F.).

CASE XXXV.—O.M. was pregnant with her first child. The indication for induction was postmaturity. Delivery occurred fifty-five hours after induction. *Bacillus welchii* was grown from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (seven pounds four ounces, twenty-one and a half inches) was stillborn. No post mortem examination was made. The mother received anti-streptococcal serum and *Bacillus welchii* antitoxin immediately after delivery. The temperature was 37.9° C. (100.2° F.) the day after delivery and on the day following.

CASE XXXVI.—E.R. was pregnant with her first child. The indication for induction was postmaturity. Delivery occurred eleven hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (five pounds thirteen ounces, nineteen inches) lived. The mother's temperature remained normal.

CASE XXXVII.—I.McN. was pregnant with her first child. The indications for induction were albuminuria and pyelitis. Delivery occurred forty-two hours after induction. Coliform organisms and non-hæmolytic streptococci were isolated from both the tissue of the placenta and from the blood of the placental vessels. The child (four pounds fourteen ounces, nineteen inches) was jaundiced on the fourth day of life and died on the fifth day. Coliform organisms were cultivated from blood obtained by heart puncture immediately after death. Similar organisms were isolated at autopsy from the spleen and from the lung. Cultures from the liver remained sterile. The mother's temperature was 37.9° C. (100.2° F.) the day before delivery.

CASE XXXVIII.—E.W. was pregnant with her first child. The indication for induction was preeclampsia. Delivery occurred thirty-four hours after induction. Coliform organisms, non-hæmolytic streptococci and an unidentified sporing anaerobe were isolated from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (three pounds fifteen ounces, seventeen

and a half inches) lived. The mother's temperature was 37.9° C. (100.2° F.) the day of delivery, and rose to 37.9° C. (100.2° F.) three days after delivery.

CASE XXXIX.—A.F. was pregnant with her second child. The indication for induction was postmaturity. Delivery occurred five hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (seven pounds six and a half ounces, twenty and a half inches) lived. The mother's temperature remained normal.

CASE XL.—M.N. was pregnant with her first child. The indication for induction was mild hydramnios at term. Delivery occurred seventeen hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (seven pounds three and a half ounces, twenty-one inches) lived. The mother's temperature rose to 38.3° C. (101° F.) the sixth day after delivery, probably from a pyelitis that was present.

CASE XLI.—M.H. was pregnant with her first child. The indication for induction was preeclampsia. Delivery occurred seventy-five hours after induction. Haemolytic streptococci and *Streptococcus viridans* were isolated from both the placental tissue and the blood of the placental vessels. The child (three pounds fourteen ounces, eighteen and three-quarter inches) was stillborn and showed signs of maceration of the lower extremities. Haemolytic streptococci and *Streptococcus viridans* were isolated from the heart blood and the spleen. Cultures from the liver and the lung remained sterile. The mother's temperature rose to 37.6° C. (99.8° F.) two days after delivery.

CASE XLII.—M.McC. was pregnant with her first child. The indication for induction was preeclampsia. Delivery occurred sixty-six hours after induction. *Staphylococcus albus*, a non-haemolytic streptococcus and coliform organisms were isolated from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (four pounds three ounces, seventeen inches) lived. The mother's temperature rose to 37.8° C. (100° F.) the day following induction, to 38.6° C. (101.6° F.) the day of delivery, to 38.9° C. (102° F.) two days after delivery, on which day a blood culture was made. The temperature was normal on the sixth day after delivery. The blood culture yielded a non-haemolytic streptococcus and a small anaerobic Gram-negative bacillus.

CASE XLIII.—E.S. was pregnant with her third child. The indication for induction was postmaturity. Delivery occurred thirty-six hours after induction. *Bacillus welchii* was cultivated from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (seven pounds thirteen and a half ounces, twenty-two inches) lived. The mother's temperature rose to 40° C. (104° F.) on the fourth and fifth days after delivery, probably owing to a pyelitis which was present.

CASE XLIV.—F.M. was pregnant with her second child. The indication for induction was albuminuria. Delivery occurred thirty-eight hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (nine pounds five ounces, twenty-two and a half inches) lived. The mother's temperature did not rise above 37.3° C. (99.2° F.).

CASE XLV.—A.C. was pregnant with her eleventh child. The indication for induction was myocarditis. Delivery occurred fifty hours after induction. An anaerobic streptococcus was isolated from the placental tissue. Cultures from the blood of the placental vessels remained sterile. The child (seven pounds four ounces, twenty inches) lived. The mother's temperature remained normal.

CASE XLVI.—N.H. was pregnant with her first child. The indication for induction was albuminuria. Delivery occurred twenty-two hours after induction. Cultures from both the placental tissue and the blood of the placental vessels remained sterile. The child (seven pounds fifteen ounces, twenty inches) lived. The mother's temperature did not rise above 37.3° C. (99.2° F.).

APPENDIX II.

Bacteriological Notes.

The coliform organisms cultivated were doubtless of intestinal origin. All strains produced acid and gas from lactose, gave a positive reaction to the methyl red test and no reaction to the Voges Proskauer test, formed indol from peptone water and failed to liquefy gelatin. Apart from the fermentation of lactose, the strains from different cases differed in their ability to ferment the various test carbohydrates, but in every instance the cultures from different sources from the same case gave identical results. Thus the four cultures in Case XVI produced acid and gas in lactose, saccharose and salicin, and failed to ferment dulcitol, while the five cultures in Case XXXVII gave acid and gas in lactose, saccharose and dulcitol, but failed to ferment both adonite and salicin.

The six strains of *Bacillus welchii* were virulent for the guinea-pig, and in each instance *Bacillus welchii* antitoxin afforded complete protection.

Of the ten strains of aerobic streptococci cultivated from the placentas of the induced cases, four were *Streptococcus viridans*, one a haemolytic streptococcus (not Group A, Lancefield,²⁰ 1933) and five produced no change in sheep blood agar plates. Aerobic streptococci were isolated from the placentas of three of the control cases; two of the strains were *Streptococcus viridans* and the third a haemolytic streptococcus (not Group A).

Anaerobic streptococci were obtained in nine of the cases in which induction was performed and in four of the controls. In spite of repeated platings, most of the strains showed both large and small cocci and chain formation was variable. When these strains were grown in meat broth containing blood, they failed to produce the peculiarly offensive odour which is usually produced by the anaerobic streptococci associated with severe puerperal sepsis.

Diphtheroid organisms, which were strict anaerobes, were cultivated from the placentas in eight of the control cases. These strains grew slowly; three to seven days' incubation was necessary before growth was detected in the original cultures. This slow growth may have been the reason why this type of organism was not recovered from the placentas in the cases in which induction was performed, at least in those cases in which the placenta was infected with aerobic organisms that might well mask the presence of slowly growing bacteria. Grown on solid media, these anaerobic diphtheroids appeared as Gram-positive rods, but when they were grown in broth, coccal forms were usually present also.

Agglutination and Agglutinin Adsorption Tests.

Agglutination and agglutinin adsorption tests were carried out with the *Bacillus coli* strains from Cases VIII, XV, XVI, XVIII, XXI, XXII, XXVI and XXXVII. In each of these cases the culture of *Bacillus coli* obtained from the placental tissue was used to produce agglutinating sera. The sera were prepared in English rabbits by the intravenous injection, at bi-weekly intervals, of increasing doses (initial dose one cubic centimetre) of twenty-four hour cultures in tryptic broth, in which the organism had been killed by heat (one hour at 60° C.).

CASE VIII.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) spleen, (d) lung. The serum prepared against strain (a) agglutinated this strain and strains (b), (c) and (d) to a titre of 1 in 2,560. Absorption of this serum with emulsions of strain (a), (b), (c) or (d) completely removed or decreased by the same amount the agglutinins for the strain used for the absorption and also for the other three strains. Thus serum absorbed with strain (b) and tested for agglutinins after absorption gave the results shown in Table VI.

CASE XV.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) spleen, (d) lung. The serum prepared against strain (a) agglutinated each of the four strains (a), (b), (c) and

TABLE VI.

Strain.	Serum Dilution.				
	1 40	1 80	1 160	1 320	1 640
(a)	++	+	+	Trace	—
(b)	++	+	Trace	Trace	—
(c)	++	+	+	+	—
(d)	++	+	+	Trace	—

(d) to a titre of 1 in 5,120. Absorption of this serum by any one of these four strains reduced the agglutinins to the same extent for all strains. After absorption with strain (d) the results obtained were as shown in Table VII.

TABLE VII.

Strain.	Serum Dilution.				
	1 40	1 80	1 160	1 320	1 640
(a)	+++	+++	++	+	—
(b)	+++	+++	++	Trace	—
(c)	+++	+++	++	Trace	—
(d)	+++	+++	+++	+	—

CASE XVI.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) liquor amnii, (d) wound. The serum prepared against strain (a) agglutinated this strain and strains (b), (c) and (d) to a titre of 1 in 5,120. Absorption of this serum by any one of these four strains reduced the agglutinins to approximately the same extent for all strains.

CASE XVIII.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) liquor amnii, (d) wound. The serum prepared against strain (a) agglutinated all four strains to a titre of from 1 in 2,560 to 1 in 5,120. Absorption of this serum by any one of these strains reduced the agglutinins to approximately the same extent for all strains.

CASE XXI.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) liquor amnii, (c) lung. The serum prepared against strain (a) agglutinated all three strains to a titre of 1 in 2,560. Absorption of this serum by any one of these three strains reduced the agglutinins to approximately the same extent for all strains. In some experiments the agglutinins were completely removed.

CASE XXII.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels. Serum prepared against strain (a) agglutinated both strains to a titre of 1 in 1,280. Absorption of this serum with either strain completely removed the agglutinins for both strains.

CASE XXVI.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) heart blood, (d) lung, (e) liver, (f) spleen. The serum prepared against strain (a) agglutinated all strains to a titre of 1 in 2,560 to 1 in 5,120. Absorption of this serum with any one of these six strains reduced the agglutinins to approximately the same extent for all strains.

CASE XXXVII.—Cultures of *Bacillus coli* were made from (a) placental tissue, (b) blood of placental vessels, (c) blood (heart puncture), (d) spleen, (e) lung. The serum prepared against strain (a) showed a low titre, although the rabbit received seven injections, ranging from 2,000 M. to 20,000 M. over a period of four weeks. The agglutination results were as shown in Table VIII.

TABLE VIII.

Culture.	Serum Dilution.						
	1 40	1 80	1 160	1 320	1 640	1 1280	1 2560
(a)	+++	+++	+++	+++	+++	+	—
(b)	+++	+++	+++	+++	++	+	—
(c)	+++	+++	+++	+++	+	Trace	—
(d)	+++	+++	+++	+++	+	+	—
(e)	+++	+++	+++	+++	++	+	—

Absorption of this serum by any one of these five strains reduced the agglutinins to approximately the same extent for all strains. After absorption with strain (e) the results obtained were as shown in Table IX.

TABLE IX.

Culture.	Serum Dilution.				
	1 40	1 80	1 160	1 320	1 640
(a)	++	+	—	—	—
(b)	++	Trace	—	—	—
(c)	++	—	—	—	—
(d)	++	Trace	—	—	—
(e)	++	Trace	—	—	—

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CYCLOPROPANE ANÆSTHESIA: A PRELIMINARY SURVEY.¹

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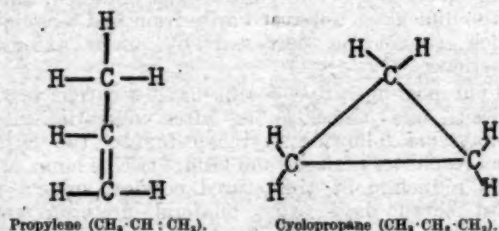
THE use of cyclopropane as a general anaesthetic is firmly established in Canada and the United States of America, and is increasing in England,

¹ Read at a meeting of the Sydney Hospital Clinical Society held on April 7, 1937.

but so far has been of only limited extent in Australia. In America it is rapidly displacing nitrous oxide and ethylene in major surgical work not only because of its greater potency, but also of the high oxygen concentrations possible, and despite its relative expensiveness. Its toxicity is insignificant in comparison with that of ether and chloroform, with which grave metabolic disturbances are apt to occur, especially in long administrations and poor-risk cases. These agents involve severe and progressive depletion of alkali and glycogen reserves⁽¹⁾ which, with cyclopropane, is practically non-existent,⁽²⁾ and while a similar advantage may be claimed for nitrous oxide or ethylene, their relatively low potency and the necessity in their successful administration for a considerable degree of anoxæmia greatly restrict their utility.

The properties of cyclopropane as an anæsthetic were first described, in 1929, by Lucas and Henderson, of Toronto, Canada.⁽³⁾ Waters and his co-workers,^{(4) (5)} of Madison, Wisconsin, United States of America, studied it extensively and proved its efficacy and safety in the human subject, with the result that in the past two years its use has spread enormously. Griffith,⁽⁶⁾ of Montreal, first used it clinically in Canada, while Sykes⁽⁷⁾ and Rowbotham⁽⁸⁾ pioneered it in England, so that there is now a wide body of competent opinion convinced as to its value. It was first used in Sydney by H. J. Daly at this Hospital in July, 1936, with excellent results for an excision of lung.

Its chemical formula is C_3H_6 , the same as that of propylene, which is the second member of the olefine or ethylene series of unsaturated hydrocarbons. Propylene, however, is an aliphatic or open-chain compound; cyclopropane is carbocyclic, all bonds of affinity being satisfied. The structural formulæ are as follows:



Much experimental work had previously been done on propylene which, while showing great promise as an anæsthetic, also produced some serious toxic effects. For these the isomer, cyclopropane, was blamed, but investigation proved it not only innocent but of the utmost safety and value, its few toxic manifestations possibly being due to propylene itself or to other contaminants not excluded in the manufacture. At the present time a product of high purity is available, and steadily expanding demand is resulting in lower prices so that even in this country it should not cost more than three shillings per gallon. Being gaseous, it is supplied compressed in small cylinders, a very convenient type being the "amplons" of Squibb and Sons, similar in principle to the carbon dioxide "sparklets" so well known here. It is

highly inflammable, and even explosive when mixed with oxygen in proportions suitable for anæsthesia,⁽⁹⁾ but in practice the mixture would probably ignite with difficulty, as it is saturated with water vapour. The closed method of administration offers further safeguards against external sparks and flames, while the risk of internal sparking from static charges is practically non-existent owing to this high humidity of the enclosed gases. Since economy and efficiency demand minimal external leakage, there is no offensive and risky contamination of the operating theatre atmosphere.

The necessity for economy and safety has resulted in the perfection of carbon dioxide absorption apparatus, and so the cost of administration approximates to that of the other gaseous anæsthetics given by current methods, while a degree of precision and control hitherto unknown is established. The closed circuit method is of outstanding merit,⁽¹⁰⁾ involving the following important considerations:

1. An atmosphere is presented to the patient composed of oxygen and cyclopropane in proportions sufficient to produce the desired depth of anæsthesia. These vary respectively from approximately 90% and 10% for light to approximately 75% and 25% for deep anæsthesia,⁽¹¹⁾ there being much individual variation and also some dilution by nitrogen already in the lungs and tissues. In practice accurate determination of percentages is unimportant; the patient's reactions are the criteria of dosage, just as they are with ether.

2. Oxygen is run in continuously in amounts sufficient to meet the patient's basal requirements, usually varying between 200 and 400 cubic centimetres per minute (in thyrotoxicosis up to 600 cubic centimetres per minute may be required).

3. By a suitable arrangement of valves and tubing this atmosphere is made to complete a circuit, being motivated by the patient's respiratory efforts.

4. Carbon dioxide is absorbed from this atmosphere by diverting it through granulated soda-lime, placed in a container which may be thrown in or out of circuit at will.

5. The enclosed gases rapidly become saturated with water vapour, reducing the danger of ignition and also the irritation arising from desiccation of mucous surfaces. Little condensation occurs in the distal parts of the apparatus, where the temperature of the gases is maintained by the heat evolved in the process of carbon dioxide absorption. Thus excessive loss of heat and fluid from the patient is prevented, an important consideration in severe and prolonged operations.

Compared with current intermittent or continuous flow methods of gas anæsthesia, the carbon dioxide absorption technique is remarkably economical. In the former the rate of flow of the gaseous mixture is largely determined by the necessity for carrying off excess carbon dioxide, and varies between five and fifteen litres per minute. In the latter, after supplying an initial ten to fifteen litres for preliminary saturation, basal supplies only of oxygen are required, together with occasional small additions of cyclopropane to make up for loss by leakage and diffusion. One, or at most two gallons of cyclopropane should

do for any duration of anaesthesia, while oxygen consumption will average about twenty litres per hour, that is, about one-sixth of the quantity required by the older method.

Cyclopropane is non-irritant and non-offensive. Unconsciousness is produced rapidly and pleasantly, without excitement. Any form of premedication may be used, but heavy doses of opium derivatives tend to hinder relaxation, and either atropine or hyoscine in moderate dosage is desirable to restrain the tendency to salivation that is otherwise likely to occur. Relaxation is usually adequate, but if not may be ensured by the addition of small quantities of ether or chloroform, the toxicity of which is minimal in the high oxygen concentrations present.⁽¹²⁾ As with all other anaesthetics, sufficient time must be allowed for adequate saturation before surgical procedures are begun. Otherwise troublesome and sometimes persistent laryngospasm is apt to occur, necessitating the passage of a Magill endotracheal tube for its correction and control. In spite of this spasm or the presence of other minor degrees of respiratory obstruction, the colour remains good, and the same applies with the shallow breathing of excessive depth. Nevertheless, it is important to have at all times a perfectly clear airway, as otherwise carbon dioxide retention occurs, resulting in an undesirable rise of the blood pressure and excessive sweating. If the attainment of adequate depth involves undue respiratory depression, the addition of ether is preferable to pushing cyclopropane.

There is a wide margin between respiratory paralysis and cardiac failure with cyclopropane. If necessary, adequate gaseous exchanges may be maintained by intermittent bag-compression over long periods and with perfect safety, while dilution of the mixture with oxygen will produce rapid lightening of the anaesthesia. Similarly, too light a level is readily and rapidly corrected by adding small amounts of cyclopropane, so that the degree of positive control available with this agent and technique makes for maximum safety.

Apart from minor disturbances of cardiac rate and rhythm, seen usually in deep anaesthesia, cyclopropane has little toxicity to the circulatory system, the compensatory sympathicotonia and hyperadrenemia of ether and chloroform anaesthesia⁽¹³⁾ being absent. This probably accounts for the increased capillary oozing seen with cyclopropane, there being no circulatory depression, and therefore no arteriolar contraction directed to the maintenance of the blood pressure. There is also little disturbance of glycogen and alkali reserves, and hyperglycaemia is uncommon.⁽¹³⁾ If carbon dioxide retention occurs from respiratory obstruction or depression, or if absorption is imperfect, a considerable rise of blood pressure will occur, but, as indicated above, this may be readily controlled. The onset of arrhythmia, bradycardia, and especially tachycardia, indicates an excessive concentration of cyclopropane, either from attempting too rapid induction or from the maintenance of profound depth. These manifestations are of no permanent significance, and disappear on reducing the concentration or on terminating the anaesthesia, but if they are unduly troublesome or

alarming, a change to oxygen-ether or nitrous oxide-oxygen-ether is indicated. Such disturbances nevertheless occur quite frequently with ether, especially in the lighter levels of anaesthesia. With chloroform, of course, they suggest the imminence of ventricular fibrillation, and in view of the present experimental status of cyclopropane the possibility of the occurrence of such a catastrophe with it should be borne in mind.⁽¹³⁾

Recovery from cyclopropane is speedy, but not so dramatic as with nitrous oxide. After-sickness is usually absent or only slight, and if repeated vomiting occurs it is often due to other factors than the anaesthesia. The incidence of post-operative complications in general is reduced,⁽¹⁴⁾ although the pre-operative condition of the patients and the selection of this anaesthetic for bad risk cases is apt to obscure this feature. Its preeminence in this regard, however, is demonstrated by the fact that it may be given without fear to patients with active acute and chronic pulmonary disease.

To date thirty-nine administrations have been completed, in twenty of which cyclopropane was the sole anaesthetic, while in a further seven some ether (never more than half an ounce) was added in the induction stage to ensure adequate relaxation. In one case a complete change to oxygen-ether was made for this reason. In four cases cyclopropane was quite satisfactory, but uncontrollable gas leakages were causing excessive consumption, so anaesthesia was continued with nitrous oxide and oxygen. In two cases nitrous oxide and oxygen was the main anaesthetic, cyclopropane being added in small quantities to provide rapid control of lightness. It was similarly used in a further three cases as an adjuvant to nitrous oxide and oxygen and ether anaesthesia. Finally, in two cases its potency was inadequate, free addition of ether and chloroform being necessary before control was obtained. Most of the difficulties appear to arise from bad technique, which should be corrected by more extensive experience.

Four post-operative deaths have occurred in this series: one three hours after operation from emphysema following thyroidectomy; the second from respiratory fatigue and failure twelve hours after open reduction of a fractured cervical spine; the third seven days after bilateral ureteral transplantation; and the fourth eight days after thoracoplasty in a case of active pulmonary tuberculosis. No death appears to be attributable to the anaesthesia, although in the first a considerable degree of respiratory obstruction in the latter part of the operation may have been a contributory factor.

The following operations were performed in the series: partial thyroidectomy, 5; tonsillectomy, 4; prostatectomy, 3; hysterectomy, 3; plastic operation on nose and face, 3; perineal repairs, 2; laparotomy, 2; ovariectomy, 2; amputation of the breast, 2. Of the following operations, one each: bilateral radical antrostomy, excision of empyema scar, excision of glands of neck, open reduction of fractured cervical spine, excision of uterine polypus, herniotomy, dental clearance, pyelolithotomy, ureteral transplantation, amputation of toes and subcutaneous

nodules, thoracoplasty, mastoidectomy, and appendicectomy and internal shortening of round ligaments.

The longest administration was for three and a quarter hours; two others were for two and one and three-quarter hours respectively; and the remainder ranged between three-quarters and one and a quarter hours. No patient showed more than slight indications of post-operative shock, excepting the patient subjected to thoracoplasty, who had serious blood loss. Of the patients subjected to thyroidectomy, two exhibited severe and one mild toxicosis; all three were in excellent shape both during and after operation.

This series of cases is too small to warrant detailed analysis, but it does serve to demonstrate the possibilities of cyclopropane and the desirability for its further investigation and trial.

My thanks are due to the various members of the honorary surgical staff of this hospital and to other surgeons who have provided me with opportunities for using this new agent and technique.

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SMALL ANEURYSMS AT THE BASE OF THE BRAIN AND SUBARACHNOID HÆMORRHAGE.¹

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SINCE 1920, in 3,670 *post mortem* examinations at the Adelaide Hospital and at the Mental Hospital, Parkside, there have been nineteen cases in which small aneurysms were present on the circle of Willis or its branches. Two of the aneurysms had not ruptured and in another case an actual aneurysm was not found, though one was probably present. There have been in addition twelve cases of pia-arachnoid hæmorrhage in which close inspection has

failed to reveal the presence of a small aneurysm which had ruptured, and five examples of subdural hæmorrhage apparently not due to trauma.

Small Aneurysms on the Circle of Willis or its Branches.

Of the sixteen examples in which the ruptured aneurysm was found, ten were in males and six were in females. The ages of the males were 20, 28, 36, 36, 49, 54, 58, 62, 67 and 75 years. The ages of the females were 20, 34, 48, 51, 61 and 62 years. The case in which an aneurysm was probably present, but was not with certainty detected, was that of a woman of 56. The two unruptured examples were in a male of 71 and a woman of 46.

Pia-Arachnoid Hæmorrhage without Aneurysm.

The patients who had pia-arachnoid hæmorrhage without any aneurysm comprised nine males, aged 19, 22, 25, 40, 43, 69, 71, 71 and 75 years, and three females, aged 33, 54 and 60 years.

Subdural Hæmorrhage not due to Trauma.

The persons with subdural hæmorrhage not due to trauma comprised males aged 42, 44, 68 and 69, and a woman aged 62. In some of the cases with a pia-arachnoid hæmorrhage, with or without the presence of an aneurysm, there has also been a little blood in the subdural space.

Not included in the above is an infective aneurysm of the middle cerebral artery, with extensive cerebral hæmorrhage and subdural clot, in a girl of nineteen, who had malignant endocarditis of the mitral valve and left auricle, old infarcts in the spleen and kidneys, and a large infective aneurysm in the anterior aspect of the abdominal aorta.

In most of the instances of pia-arachnoid hæmorrhage at the base of the brain, secondary to leakage from an aneurysm, the extravasation of blood has been bilateral. In the case under special review, that of a lad of twenty, the extravasation of blood was confined mostly to one side, and this was readily explainable by the aneurysm's being situated some distance from the circle of Willis on the first branch of the middle cerebral artery.

In one case, that of a woman of twenty, the aneurysm was so situated as to occupy the position of a cerebello-pontine angle tumour, for which the condition was very reasonably mistaken. It was only at autopsy that its true nature was discovered.

When rupture of these small aneurysms occurs and when the rupture is on the side of the aneurysm that is applied to the brain tissue, the escaping blood will sometimes be directed into the brain tissue rather than into the meshes of the pia-arachnoid. In this way in some instances the lateral ventricles have been filled with blood.

Associated Lesions.

Pia-Arachnoid Hæmorrhages with Small Aneurysms.

Amongst the eighteen patients in whom aneurysms were found, the following associated conditions were met with. Two patients, a man of

¹A summary prepared for a demonstration and discussion on a case of subarachnoid hæmorrhage at the Adelaide Hospital, 1936.

54 and a woman of 61, were in the mental hospital suffering from melancholia. It may be reasonably surmised that the aneurysms may have played a part in the development of this mental condition. In the man there were two aneurysms, one the size of a walnut, so that considerable disturbance, vascular and from pressure, might be expected. A man of 62 had a healed dissecting aneurysm of the right common iliac artery, which may indicate a general tendency to a defect in the walls of the arteries. Hypertrophied hearts with renal fibrosis were met with in three patients, men aged 67 and 75 years, and a woman aged 34 years; and hypertrophied hearts alone in two men, aged 36 and 54 years. It will be noted that two of these patients were relatively young, only 34 and 36 years of age, and it is possible that the high blood pressure which was presumably associated with the hypertrophy of the heart may have played a part, if not in the development of the aneurysm, at least in its rupture. In one case, that of a male of 49, special mention is made of atheroma of the vessels at the base of the brain. A woman of 46 had infective nephritis, and a man of 71 had a carcinoma of the stomach and hæmorrhagic pyelitis; but it is impossible to see any special connexion between these conditions and the aneurysms. In only three instances were Wassermann tests carried out; two of these gave negative results, in men of 28 and 36, and one gave a positive result with the cerebrospinal fluid, namely, in the man just mentioned, aged 71 years, with carcinoma of the stomach and hæmorrhagic pyelitis.

Pia-Arachnoid Hemorrhages without Small Aneurysms.

Atheroma of the vessels at the base of the brain was present in five of the twelve persons, namely, in men aged 69, 71 and 75 years, and two women of 54 and 60.

In the man aged 75 years there was rupture of the anterior communicating vessel, and in the woman of 60, extravasation of blood all over the parietal lobe.

A lad of 19 was a diabetic who had a systolic blood pressure of 190 and a diastolic pressure of 90 millimetres of mercury.

The man of 22 had abscesses in the prostate and left kidney. This man also had punched-out ulcers of the stomach, and another patient, a man of 43, showed an old ulcer of the pylorus.

The man of 40 had *otitis media*, for which an operation was performed, and at the *post mortem* examination pia-arachnoid hæmorrhage was found over the cerebellum and between its leaves, with some subdural blood extravasation as well.

Subdural Hemorrhages Apparently Unconnected with Trauma.

In a man, aged 42 years, a subdural hæmorrhage was a feature in atypical lymphatic leucæmia.

A man, aged 44 years, had had pains in the back of the head and neck for three days before death without any history of trauma.

A woman of 62 had a ruptured vessel in the left mid-parietal region.

A man of 68, suffering from senile dementia, had much atheroma of the vessels at the base of the brain; one of the vessels had ruptured.

A man of 69, who was comatose and who had sugar and albumin in the urine, was found to have an hypertrophied heart and renal fibrosis with, apparently, rupture of one of the emissary cerebral veins.

It will be seen from the above that the three persons aged over 60 years had apparently ruptured vessels from which hæmorrhage had taken place into the subdural space.

AN INTERESTING SELF-PROTECTING MECHANISM IN THE PROTOZOAN VORTICELLA.

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As I have never encountered in text-book or other communication any reference to the following observation, and as it is easily carried out and illustrates several vital general biological conceptions, I think it worthy of record.

One of the simplest definitions of a living organism may be stated thus: a living plant or animal is an assimilatory, reproductive, self-protective mechanism found only in relation to cellular structure. All these qualities are uniquely characteristic of living things and are not found in non-living matter.

Growth, sensitiveness, conduction and movement are frequently found outside living organisms. The self-protective nature of all that lives and continues to live is especially important in the maintenance of individual or species from amœba to man.

The Vorticella is a common protozoan easily cultivated in hay infusions. It is a bell-shaped animal attached to some floating or attached material by a fine contractile stalk.

When the animal is undisturbed, its stalk is relaxed and elongated, and at the free end of the bell a spiral circle of cilia sweeps little particles of food-stuff into a little gullet ending in the protoplasm of the creature's interior.

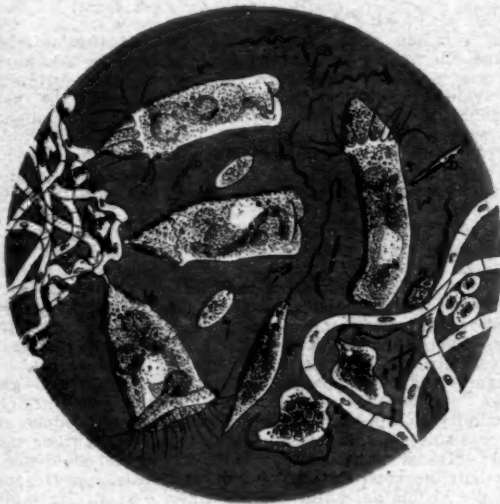
When it is disturbed, a central "muscular" fibril in the stalk contracts, and as the ciliary disk closes, the little animal is pulled out of danger.

The present observation occurred when a preparation of Vorticella was put up before dinner one evening with the object of displaying some to an interested friend. There were under the field forty Vorticellæ actively feeding. To my surprise, some hour or half-hour later, when my friend arrived, there was not a single Vorticella visible.

A search of the slide, however, showed a number of free protozoans as figured in the drawing (V¹).

These were eventually recognized as Vorticella bells with the feeding disk closed and swimming by cilia that had grown around the basal portion and were proceeding in an aimless way base first.

Further observations showed that in from half to one hour from setting up the preparation the animal closed its feeding disk, and around its base, close to the stalk, a bright line appeared, which became wavy and eventually became drawn out into thick cilia. Shortly after this the animal snapped itself off the stem and became the free form above described.



It naturally occurred to me that the whole complex process was most likely related to an attempt to escape from unsuitable conditions.

Two things had happened on my slide: the oxygen content of the preparation was diminishing and the excretory waste products were increasing.

This state of affairs must happen frequently under natural conditions, and unless there was a way of escape to better circumstances the animal and the race would die.

I pictured the drying-up edge of a pool and the same process occurring: the moving away from the poisonous surrounding, feeding disk closed to prevent further poisoning, the migration to a better spot, the growing of a new stalk and resumption of its active metabolic life.

Very simply I was able to prove my hypothesis. I simply took the preparation containing the free forms, and by means of a pipette at one end and blotting paper at the other I removed the old and gave the animals fresh oxygenated water.

Immediately a change in the nature of the aimless wandering of those backwards swimming creatures took place. Their movement became, if we may call it so, purposeful. The animals, with strong lashing of their basal cilia, became applied to some little bit of weed or *débris* and soon a bright spot

appeared at the extreme base, the beginning of a new stem. Progressively the basal cilia became irregularities, then a wavy line, and disappeared, and the feeding disk opened anew. Gradually the stem grew and the animal returned to the original active form. It seemed to me a pretty instance of the self-protective mechanism and worth this short record, even if perchance somewhere it has been noted before.

The drawing is a composite one made by myself from the actual preparation.

The growth of basal cilia, of course, is a well-known phenomenon in relation to various reproductive phases. The interest of the present observation is the utilization of the growth of basal cilia to surmount a change in surroundings.

There is also food for thought in the, shall we say, deliberate closing of the feeding disk and the reverse movement of the animal, quite distinct from the mode of progression of accidentally broken off bells, which swim disk open and disk forward.

Reviews.

AN INTRODUCTION TO GENERAL PRACTICE.

SIR E. KAYE LE FLEMING'S small book, "An Introduction to General Practice" will be invaluable to English students and graduates in medicine who intend to engage in general practice, and much of the valuable advice which the distinguished author gives to his junior colleagues overseas is equally applicable to the conditions of medical life in Australia.¹ The writer is not greatly concerned with the scientific aspects of the profession, but lays great stress upon the necessity for acquiring those qualities of mind and heart which make a doctor acceptable to the common run of his patients, but which are not readily learnt in the impersonal atmosphere of a teaching hospital. Most patients, the author points out, are ready to take a doctor's professional abilities for granted; their keenest criticism is concerned with his method of handling his patients, of his possession or lack of all that is summed up in the phrase "bedside manner".

Sir E. Le Kaye Fleming has something of interest to say on the subject of the unorthodox practitioner. Should the doctor take any active steps to end a state of affairs which forbids an unqualified dentist to pull teeth, but which permits any unregistered practitioner with impunity to attend to the rest of the body? In the field of manipulative surgery the orthopaedic surgeon has at length ousted the "bone-setter" from the domain which the neglect of qualified practitioners permitted him so long to occupy. In these matters the public, by reason of its credulity and love of mysticism, receives the treatment it demands; the doctor at this stage will do well to realize that more is to be gained by adopting an attitude of sympathy to his patient than by opposition, since nothing will prevent the patient from seeking the advice upon which his mind is set.

Another important problem upon which Fleming writes wisely is the encroachment of large general hospitals upon what were formerly regarded as the rightful spheres of activity of the general practitioner. In England, as in Australia, this tendency is increasing greatly; and no one of us is so foolish as to maintain that the rapid march of medical science permits the average practitioner

¹ "An Introduction to General Practice", by E. Kaye Le Fleming, M.A., M.D.; 1936. London: Edward Arnold and Company. Crown 8vo, pp. 150. Price: 5s. net.

to maintain a high standard of skill in an ever-widening diversity of subjects. More and more, as an instance, the domiciliary management of midwifery is on the wane, and the example of midwifery is but one amongst many. There is lacking a strong link between the hospital and the ordinary doctor, but no health service can claim perfection which does not provide such a bond. Perhaps the remedy lies in the extension of the cottage hospital principle or in the establishment of wards at the large hospitals, wherein private practitioners may attend their own patients.

MEDICAL PSYCHOLOGY.

IN "Modern Discoveries in Medical Psychology" Dr. Clifford Allen gives an exposition of some of the more prominent methods of psychological treatment, from that of Mesmer onwards.¹ It would appear that he has developed along more popular lines a theme dealt with in 1926 by Bernard Hart in his Goulstonian Lectures on "The Development of Psycho-Pathology". In discussing hypnosis, Dr. Allen writes: "If only chloroform had not been invented for twenty years, it is possible that it would never have been needed." This is only one amongst many uncritical observations which detract from the scientific worth of the book, and which appear to have been made to attract the general rather than the medical reader.

From Mesmer Dr. Allen proceeds to Janet and Morton Prince, whose famous "Sally" case, with multiple personalities, is described at length.

The Freudian teaching is described fully, and it would appear adequately, in fairly simple language. It is a pity that Dr. Allen is not more careful about the terms "analysis" and "psycho-analysis". While Freud created and elaborated the method of mental exploration which goes by the name of "psycho-analysis", the analysis of mental processes was carried out long before Freud, in the form of dream interpretations and detailed inquiry into the patient's memories. The statement, therefore, that analysis was Freud's own invention is less exact than one would expect from a work of this sort. Later on, in regard to Freud's theory, Dr. Allen states: "It would not be extravagant to suggest that the freer and healthier mode of modern life, with its franker moral code, its emphasis on the exposure of the body to sun and air, and its freedom of speech and conduct, is the result of Freud's teaching." We seem to recall that a similar ideal of living was followed centuries ago in the golden age of Grecian culture.

After Freud we come to Adler and Jung. Dr. Allen might have mentioned at least one of Adler's distinguished disciples in the shape of the late Dr. Crookshank, who did a great deal towards applying the Adlerian theory of "organ jargon", especially since, as the author admits, Adler himself added very little to the principles which he propounded in 1913. Incidentally, miner's nystagmus occurs in a minority of those "who have had some great shock when working underground". While admitting many valuable applications of Adler's teaching to the treatment of disorders of conduct in adults and children, we can hardly subscribe to Dr. Allen's statement: "For his understanding of children and development of their therapy we must be grateful to Adler, and possibly the Child Guidance Clinic will be a permanent memorial to his fame for all time", since the child guidance clinic as generally understood is the direct development of the "team" method applied by Healy in Chicago and later in Boston to the treatment of delinquent children.

Jung's thinking, feeling, sensational and intuitive types are described with moderate clarity, but perhaps only Jung himself has a full appreciation of their characteristics.

Dr. Allen shows a strong bias against the handling of neuroses by ministers of religion, and his own conception

of religion appears to be narrow and even antiquated. Anyhow, we can hardly agree that religion is a matter exclusively of the conscious mind.

The Greek on page 202 is meaningless in its present spelling, and the Latin is ungrammatical.

Kretschmer is introduced, after the manner of a well-known radio announcer, as "a man of middle height, shy in manner, and rather of the petit bourgeois type".

The final chapter is devoted to the work of Pavlov.

The book is intended for the general practitioner and medical student, and also as an introduction for those reading for higher degrees.

LATENT SYPHILIS.

IN common with many other investigators, Griffith Evans, the author of "Latent Syphilis and the Autonomic Nervous System",¹ falls into the error of over-estimating the value of clinical findings, and exhibits the usual tendency to formulate an hypothesis which is applied to too wide a variety of conditions.

From time to time undue emphasis has been placed on the part played by focal sepsis, angiospasm, infantile sexuality and other factors in the production of certain disorders; but a dispassionate survey by impartial observers has always shown that enthusiasm has obscured judgement. The same may be said of the work under review.

Acting on the assumption that latent syphilis is largely a disease of the lymphatic system, Griffith Evans proceeds to show that many conditions must, *ipso facto*, arise from this. Even allowing that his premise is sound, it does not necessarily follow that his inferences are correct. He suggests that the adenitis of chronic syphilis will affect those sympathetic nerve fibres which pass in close proximity to the lymphatic glands and thus produce such diverse states as asthma and *pes cavus*. The author states that: "It is so reasonable on pathological evidence to attribute nervous dysphagia and dyspepsia, spastic colon, chronic abdominal pain, and the like, to syphilitic infiltration of the sympathetic system." Such a statement affords an excellent example of over-compensation, for, in an attempt to correct what he regards as under-valuation of symptoms, he goes to the opposite extreme and attaches too much importance to them. He considers significant a response to the administration of mercury and iodides and the presence of a master mariner in the family, whereas a negative response to the Wassermann test, the absence of gross lesions and demonstrable spirochaetes and other clinical signs are ignored. In any case, the inference that mediastinal glands must be enlarged because the superficial glands are palpable is not sound, when consideration is given to the multiplicity of causes that may give rise to such an enlargement. It has by no means been conclusively proved that chronic syphilis is essentially lymphophilic in nature. Until this has been done it seems that the author has a very weak case.

In addition to those mentioned, the conditions which are regarded as manifestations of chronic or latent syphilis are hyperthyroidism, asthmas, angioneurotic edema, purpura, achalasia, hyperacidity, chronic constipation and many other clinical entities. Characteristic of the involved reasoning that is to be found throughout this work is the assumption that, because some authorities state that the precancerous stage consists of proliferation of the endothelium of the lymphatics, syphilis must play some part also in this, as it is also a lymphatic disease.

Too much importance is attached to purely empirical treatment and too little to clinical and pathological findings. If the author's assertions were accepted, the physician and the surgeon might well retire and leave the field to the specialist in the treatment of syphilis.

¹"Modern Discoveries in Medical Psychology", by C. Allen, M.D., M.R.C.P., D.P.M.: 1937. London and Melbourne: Macmillan and Company Limited. Demy 8vo, pp. 290. Price: 8s. 6d. net.

¹"Latent Syphilis and the Autonomic Nervous System", by G. Evans, M.A., D.M., F.R.C.S., D.O.M.S.: Second Edition: 1937. Bristol: John Wright and Sons Limited. Demy 8vo, pp. 168, with illustrations. Price: 7s. 6d. net.

The Medical Journal of Australia

SATURDAY, JULY 24, 1937.

All articles submitted for publication in this journal should be typed with double or treble spacing. Carbon copies should not be sent. Authors are requested to avoid the use of abbreviations and not to underline either words or phrases.

References to articles and books should be carefully checked. In a reference the following information should be given without abbreviation: Initials of author, surname of author, full title of article, name of journal, volume, full date (month, day and year), number of the first page of the article. If a reference is made to an abstract of a paper, the name of the original journal, together with that of the journal in which the abstract has appeared, should be given with full date in each instance.

Authors who are not accustomed to preparing drawings or photographic prints for reproduction, are invited to seek the advice of the Editor.

ASYMPTOMATIC NEURO-SYPHILIS.

THE United States Public Health Service regularly issues a series of brochures dealing with venereal disease, that are intended for use in the cooperative work of the service with the State health departments. The issue for March, 1937, contains a special article on asymptomatic neuro-syphilis that calls for special attention. The article is a clinical study issued from the syphilis clinics of the Mayo Clinic, the Western Reserve University, the Johns Hopkins University, the University of Pennsylvania and the University of Michigan; these clinics have been assisted by the United States Public Health Service and have received the financial support of an anonymous donor.

Asymptomatic neuro-syphilis is that type of neuro-syphilis in which the cerebro-spinal fluid gives a positive response to the Wassermann test, but in which there are neither subjective nor objective symptoms of involvement of the central nervous system. Admittedly neuro-syphilis may never give rise to symptoms, but it is in these circumstances that many of the most severe cerebral manifestations of syphilis subsequently appear. The patients

on whom the present study is based were taken from a series of 75,000 persons suffering from syphilis in all its manifestations, who sought treatment in one or other of the five cooperating clinics. Only patients who had been observed for at least two years were included, and those whose cerebro-spinal fluid had been examined on one or more occasions numbered 5,293. The cerebro-spinal fluid of 46.6% of these 5,293 patients was always "negative"; in 39.9% the report was positive and there were associated clinical signs of neuro-syphilis; in 13.5%, or 712 cases, asymptomatic neuro-syphilis was present. It was found that the characteristics of the cerebro-spinal fluid in asymptomatic neuro-syphilis were significant, and the observers were able to classify the abnormalities into four groups. As a result of this classification it was possible to determine the prognosis and to estimate in advance fairly accurately the type, the duration and the intensity of the treatment indicated. In Group I, designated mild, the lymphocyte count was ten cells or more, the globulin test might or might not give a positive reaction, and the Wassermann and colloidal tests gave no reaction. Cases in Group II were those in which the cell count and globulin content were normal or increased, the Wassermann test gave a positive reaction, and the colloidal test gave a result that was indeterminate or positive. If the reaction to the colloidal test was positive, it was usually of the syphilitic zone type. Group III cases, moderately severe, were those in which the cell count was thirty or more, the Wassermann test gave a "++++" reaction, and the colloidal curve was either normal or of the syphilitic zone type. In Group IV cases, those which were severe, the spinal fluid showed the so-called parietic formula, that is, there was considerable excess of globulin, the Wassermann test gave a strongly positive reaction, and the curve of the colloidal test was of the parietic type. The cell count was above normal, and, in addition to small lymphocytes, large lymphocytes and polymorphonuclear cells were present.

The whole report is full of interesting data; the details will be read by all who have to do with the management of clinics for the treatment of persons

with venereal disease, particularly by those working in public health departments. For the clinician there are two messages. The first is that the earlier in the course of the disease treatment is started, the lower will be the incidence of neuro-syphilis. The second is that asymptomatic neuro-syphilis must always be suspected, even if all clinical manifestations have disappeared, and even if the blood no longer yields a reaction to a specific test. The groups into which the 712 patients in the American series were divided have been set out in detail in order to show the enormous variation that can occur in the cerebro-spinal fluid when to all appearances the patient is normal. It is important to note that in spite of treatment a patient with asymptomatic neuro-syphilis may progress to a stage at which he manifests clinical signs of the involvement of his central nervous system. Of the 712 patients, 43, or 6%, developed clinical forms of neuro-syphilis; in twelve the condition was *dementia paralytica*, in ten it was meningo-vascular syphilis, and in nine it was *tabes dorsalis*. The majority of these progressions appeared by the fifth year of treatment. Another disquieting fact is that of 364 patients, three developed clinical signs of neuro-syphilis after the spinal fluid had become normal, and fourteen showed progression in other manifestations of syphilis, such as cardio-vascular disease. Of the three patients who manifested signs of neuro-syphilis after their fluid had become normal, one developed *tabes dorsalis*, another taboparesia, and the third showed signs of meningo-vascular neuro-syphilis. The explanation of these progressions is somewhat difficult, but whatever the explanation may be, it is obvious that the clinician must always be most guarded in giving a prognosis in any case of syphilis.

Current Comment.

DIET AS A FACTOR IN CAUSING NEPHRITIS.

CONSIDERABLE work has been carried out on experimental nephritis, and not a little of the earlier investigation of this subject concerned the possibility of diet as a factor in causing permanent renal disease. There were, however, many fallacies in this

early work, for some of it was done before the significance of vitamins was understood, and the diets used for the animals under experiment were deficient in the accessory food substances. Moreover, the animals chosen were not always suitable, for it is necessary for a test animal in these particular experiments to be accustomed to foods similar to those employed; otherwise it is impossible even to attempt any practical application of the results. N. R. Blatherwick and E. M. Medlar have recently described experiments in which they produced nephritis in rats by means of diets high in protein.¹ They selected the rat as being a carnivore, and followed up the work of Newburgh and Curtis, who found that severe renal damage was produced in rats fed on diets containing large quantities of beef, liver, and to a less extent beef muscle. Blatherwick and Medlar took great care with the experimental details of their work, employing animals from a standard stock, housed and looked after under ideal conditions. Specimens of urine were collected by keeping the animals in metabolism cages at the required periods, and the usual analyses were made. The obviously sick animals were killed with "Sodium amytal" and the heart blood subjected to chemical analysis to demonstrate the degree of nitrogen retention, if any, the ratio of albumin to globulin, and the cholesterol content of the blood. Careful histological studies were also made by an independent pathologist who knew nothing of the history of the animal whose organs he was examining. It was not assumed in any case that the finding of protein or casts in the urine proved the existence of nephritis, the final verdict being given only by the microscopic examination of the kidney, the blood chemistry findings being confirmatory. Other organs were also found to be affected, such as the aorta, which was often observed to be calcified, but no correlation was worked out between the renal and extrarenal changes. Many of the animals were subjected to a preliminary nephrectomy, which, as might be expected, increased considerably the liability and severity of nephritis. It is curious that the male animals were more susceptible than the females, and death also occurred earlier in the male rats. This observation is probably another instance of that familiar sex difference in the incidence of many diseases in man, a feature which is well known, but whose reason is obscure. It was found that fatty infiltration of the liver occurred in many of the animals, owing apparently to the high cholesterol content of the liver diet used.

The authors draw one conclusion from their work which suggests a parallel in human nephritis: they believe that functional derangement of the kidney antedates considerably the appearance of the frank histological changes typical of nephritis; but it was their experience that once this threshold was passed the disease developed rapidly. The general conclusions reached in this work may be summarized as follows. Chronic nephritis has been produced in

¹ Archives of Internal Medicine, April, 1937.

rats by feeding them on a diet very rich in protein. The earliest changes observed were in the urine, which contained protein and casts; but these could not certainly be said to be due to nephritis until urea retention and hypercholesteræmia were observed. How the diet produced these results is not known. It is suggested by the authors that abnormally complex metabolic processes may lead to formation of excessive or unusual end-products, and thus to strain on the glomerular mechanism, related either to excretion or retention of such substances. It is evident from reading of the curious evil side-effects that were observed when the diets were varied by sundry additions or subtractions, that extreme caution must be used both in making such experiments and in interpreting them. However, this carefully controlled work seems to have established that a high protein diet of a special kind can produce nephritis in certain animals. Whether the morbid process is an expression of overwork or of toxic spoiling by some actual or potential tissue poison is open to argument. In spite of the sureness of many popular writers on the subject, it is necessary to keep an unbiased mind on the subject of dietetics, about which accurate knowledge is only beginning to accumulate; but there is no doubt that it will prove of increasing importance in the prevention of disease.

HISTAMINE AND APPENDICITIS.

HANS SELYE has written an article on experimental production and prevention of appendicitis which will provoke much thought and consideration.¹ He remarks that it has been shown that intraperitoneal administration of a locally irritating solution (for example formaldehyde) will, in rats, cause localized appendicular lesions. The lesions are always seen in that part of the caecum of the rat which corresponds to the human appendix, and they develop from the mucosa outwards and not from the serosa inwards. It has accordingly been assumed that the lesions are not attributable solely to the local action of the irritant. It had been concluded that any non-specific stimulus which inflicts severe damage on the organism will elicit a typical reaction, which has been called an "alarm reaction", and probably some of the symptoms of such reaction are due to liberation of histamine or some kindred compounds from the bodily tissues. It seemed possible that the appendicular lesions might be evidence of a very acute, non-specific injury, such as would result from intraperitoneal injection of formaldehyde. To test this conjecture, thirty milligrammes of histamine were given intravenously to hooded black and white adult rats, and such treatment was always followed by manifestations of acute appendicitis. A *post mortem* examination seven hours after the injection revealed a very acute hæmorrhagic, phlegmonous appendicitis.

Selye frankly admits that there is no present evidence that acute human appendicitis is produced in a similar manner; but he considers that the evidence is conclusive that intravenous injection of histamine, which is a physiological body constituent, may cause localized lesions in the appendix.

When a less acute alarm reaction is evoked in animals, as, for example, by injections of formaldehyde or other noxious substances, appendicitis does not result, although other anatomical changes occur. Possibly only very sudden and severe damage would elicit a reaction in the appendix. In rats typical appendicular lesions will result if the animals are forced to exhaust themselves very rapidly. More gradually exhausting muscular exercise will cause a typical alarm reaction, but not appendicitis. Pleuritis and appendicitis are often in clinical combination. Accordingly observations were made as to whether intense irritation of the pleura would also cause appendicitis. Six adult hooded rats were given intrapleural injections of 0.25 cubic centimetre of 4% formaldehyde. They were destroyed twelve hours later, and three of them gave evidence of acute appendicitis. Possibly, therefore, in the rat, pleural irritation may cause lesions of the appendix. Appendicitis was not observed in less acute cases of general damage, that is, in the more typical form of alarm reaction. It was therefore considered whether histamine might not exert its effect on the appendix as a gradient-potential poison (*Potenzialgift*). Possibly, Selye considers, the effect on the appendix is not due to the actual concentration of histamine in the body at any time, but to the rate at which histamine concentration increases. If this were the case, it could be readily understood why appendicitis is not observed in a more chronic alarm reaction, even when other manifestations are well marked. Further, it should be possible to prevent experimental appendicitis by pre-treatment with small doses of histamine or by causing a less acute alarm reaction prior to the administration of an amount of histamine which would have evoked appendicular lesions. Selye claims that such an assumption was confirmed by experiments on hooded black and white rats, which he describes. He emphasizes the fact that nothing is known as to the actual mechanism by which histamine selectively damages the appendix or by which this effect is inhibited.

These observations of Selye need to be examined very closely and confirmed before they can be accepted.

THE ADELAIDE CONGRESS.

THE attention of the medical profession throughout Australia is centred in Adelaide, where in five weeks' time the fifth session of the Australasian Medical Congress (British Medical Association) is to be held. We wish to remind readers once more that it is not too late to become a member of what promises to be a most interesting and happy gathering.

¹ The Canadian Medical Association Journal, May, 1937.

Abstracts from Current Medical Literature.

MORBID ANATOMY.

Rhabdomyoma and Myoblastoma.

D. F. CAPPELL AND G. L. MONTGOMERY (*The Journal of Pathology and Bacteriology*, May, 1937) classify striated muscle tumours into two groups, each comprising simple and malignant types; these are rhabdomyomata and myoblastomata. Rhabdomyomata are tumours in which a proportion of cells show unequivocal longitudinal and transverse striation. Myoblastomata are tumours whose cells morphologically and tinctorially resemble muscle cells, but which are devoid of transverse striation. The authors describe two tumours of the first and four of the second type. Two neoplasms occurring in the soft palate were typical malignant rhabdomyomata. They pursued a similar clinical course, characterized by repeated local recurrences over some years and terminating with widespread dissemination by lymphatics and blood stream. Of the four myoblastomata, two occurring in the urinary bladder and one occurring in the spermatic cord appeared to be non-malignant, but one situated in the tongue presented malignant characters and proved fatal. Striated muscle tumours growing from a mucous surface tend to show a coarsely polypoid structure with broad clubbed processes, and this appearance is sufficiently characteristic to be of value in clinical diagnosis. Striated muscle tumours are locally destructive, but may also metastasize, usually first by the lymphatic pathway, but later also by the blood stream. The authors collect from the literature records of twenty-two metastasizing rhabdomyomata; these include their own two cases.

Silicious Dust Cirrhosis.

F. W. SIMSON (*The Journal of Pathology and Bacteriology*, May, 1937) has tried to produce experimental silicious dust cirrhosis in rabbits. He found that no distinctive fibrotic lesions were produced in the liver and lymph glands of rabbits following the intravenous injection of silicious dust extracted from silicotic lungs by the nitric acid method. Lesions were readily produced, however, by the intravenous injection of unaltered fine particle fractions of airborne mine dust collected in the Witwatersrand gold mines. The lesions produced affected only the organs provided with sinusoids, such as spleen, liver and lymph glands. The failure of the author to produce characteristic fibrotic lesions by the intravenous injection of mineral dust extracted from lung tissue is put down by him to one of several causes: (a)

the severe chemical treatment and excessive heating to which the dust was subjected in the process of extraction; (b) the possibility that the silicious particles had become coated with an inert insoluble substance which prevented their subsequent solution in the body fluids; (c) the possibility that the active fraction of the dust had already been removed after solution in the body, being used up in the original production of the silicotic lesions in the lung tissue.

Ligation of the Hepatic Ducts.

H. L. STEWART, A. CANTAROW AND D. R. MORGAN (*Archives of Pathology*, May, 1937) have studied the changes in the liver of the cat following ligation of a single hepatic duct. The changes in obstructed portions of the liver are with few exceptions essentially similar to those which result from ligation of the common bile duct. All are, however, much less pronounced and much less rapidly progressive. The chief additional points of difference are as follow: (a) the cirrhosis is almost purely monolobular; (b) focal necroses occur less frequently than in total stasis and develop at a later period; (c) the hepatic cells at the periphery of the lobules undergo a rather distinctive change, their cytoplasm becoming smooth and deep-staining and containing little or no stainable lipid, even when the remainder of the lobule shows large quantities; (d) lobular atrophy progresses inward from the periphery; (e) repair and regeneration of hepatic cells occur throughout periods of stasis up to sixty-two days; mitotic figures are most numerous between the twelfth and thirty-fifth days; (f) there is no essential difference in the amounts of stainable lipid in obstructed and non-obstructed lobes with periods up to sixty-two days; (g) demonstrable impairment of hepatic function may occur occasionally following ligation of single hepatic ducts.

Fat Embolism.

A. J. WATSON (*The British Journal of Surgery*, April, 1937) reviews the literature of fat embolism and reports a case. In the case reported by the author the patient was a man, aged twenty-two years, who sustained a compound fracture of the right tibia. Operation was performed, but after a period of feverishness the patient became unconscious and died. Autopsy revealed a clean fracture with no sign of local infection. The lungs showed pronounced congestion with patches of hemorrhage beneath the visceral pleura. Many punctate hemorrhages were present in the white matter of the brain, but there was no evidence of injury to the skull or of cerebral contusion or laceration. Sections of many organs stained with osmic acid showed the presence of fat embolism. The author concludes that fat emboli may be demonstrated in small numbers in about 14% of all *post mortem* examinations. In injuries, particularly in

fractures, a large degree of fat embolism may be found in the lungs and in the brain. In a small proportion of cases this produces inflammatory changes which may prove fatal. The fat globules producing the embolism probably come from the site of injury, although the precise mechanism of absorption is not known. The possibility of normal fat being a source of the emboli has been considered in the light of the experiments of Lehmann and Moore. The symptoms and signs of fat embolism fall into two main groups: pulmonary and systemic. The diagnosis of fat embolism is made mainly on the clinical progress and by exclusion of other complications of injury. Apart from this, the presence of fat in the urine and in the sputum, and the occurrence of a petechial hemorrhagic rash are important diagnostic signs. Treatment of the established condition is unsatisfactory, as no method so far tried has been successful in dislodging or breaking up the fatty globules in the capillaries.

Cerebral Lesions in Hypoglycæmia.

A. B. BAKER AND N. H. LUFKIN (*Archives of Pathology*, February, 1937) report the discovery in three cases of hypoglycæmia of numerous new and old cerebral hemorrhages. These appeared to be most numerous in the brains of those patients who had had the most severe convulsive seizures. Alterations were also found in the nerve cells. In order to eliminate the suspicion of autolysis an attempt was made to produce hypoglycæmic convulsions in rabbits and to study the brain tissue. In the brains of the rabbits no cell alteration of pathological significance was discovered.

Splenomegaly with Myeloid Transformation.

G. R. TUDHOPE (*The Journal of Pathology and Bacteriology*, January, 1937) reports a case of gross splenomegaly associated with myeloid transformation of the spleen pulp. The patient, a man of fifty-six years, was admitted to hospital because of severe attacks of flatulence and abdominal distension. A moderate microcytic anemia and a slight leucocytosis were found. Laparotomy was undertaken and an abdominal mass, previously undiagnosed, was found to be a greatly enlarged spleen. A piece of the spleen was removed for biopsy. The patient died of hypostatic pneumonia two days later. The author gives a full account of the *post mortem* findings. The most striking feature in the spleen was the presence of a large number of dark purple-red, more or less spherical nodules, varying in size up to about eighteen millimetres in diameter. On microscopic examination these areas were found to consist of a highly cellular and hemorrhagic tissue of myeloid character. The author points out that the most noteworthy feature of the case, apart

from the unusual degree of myeloid transformation in the spleen, was that, although *post mortem* examination revealed anatomical changes in the spleen, liver and marrow such as are usually associated with leuco-erythroblastosis, the changes in the blood were not diagnostic and gave no indication of the nature of the condition present. The article is illustrated by a series of photomicrographs.

MORPHOLOGY.

Innervation of the Periodontal Membrane of the Cat.

W. LEWINSKY AND D. STEWART (*Journal of Anatomy*, January, 1937) describe the innervation of the periodontal membrane of the cat. This is from two sources: (a) fibres running from the apical region, and (b) fibres entering laterally from the alveolar plates. The latter divide into two fasciculi, those running towards the apex and those running towards the gingival margin. The alveolar nerve fibres appear to form a greater proportion of the nerve fibres to the periodontal membrane than has hitherto been believed. The nerve fibres in the periodontal membrane are of two types: (a) thick fibres confined to the peripheral part of the membrane, which have specialized end organs at their termination, (b) finer nerve fibres, which pass to the deeper part of the periodontal membrane and break up into fine arborisations without terminal organs. It is suggested that the thick fibres, with their specialized end organs, are associated with tactile and pressure sensations, while the finer fibres are associated with those of pain. The authors were unable to trace any fibres into the cementum.

Bone and Tooth Development.

HANS GRUNBERG (*Journal of Anatomy*, January, 1937) discusses whether and to what extent the anomalies in the shape of teeth can be explained in terms of the peculiarities already well established in the grey-lethal mouse. The bearing of these facts on the general conception of the action of exogenous stimuli and the response of bones is considered. It is well known that bone responds to pressure by absorption. In the case of a socket the stimulus for absorption is known to be the growing tooth germ. If that germ grows vigorously, but the socket does not respond to its proper stimulus, it is evident that the lack of secondary bone absorption is due to the inability of the bone to respond, not to the absence of the stimulus. If the bone is to respond to pressure by absorption, it must be able to do so. The ability of bone to react, however, is evidently rooted in hereditary factors. If these factors act normally, as is the case in all known animals with the exception of the grey-lethal mouse,

the relationship between pressure and absorption appears to be direct, and the size and shape of the socket is determined by the growth tendencies of the tooth germ. If, however, this hereditary mechanism is upset, as in the grey-lethal mouse, the stimulus by itself is unable to induce absorption in the bone, and the size and shape of the tooth are limited and influenced by the size and shape of the socket at the time that the tooth becomes enclosed in it. From this the author concludes that bone reacts by absorption to pressure only if the hereditary basis for doing so is undisturbed. The relation between stimulus and response is therefore not direct, but subject to conditions based in the hereditary constitution of an animal.

The Infraumbilical Abdominal Wall.

GEORGE M. WYBURN (*Journal of Anatomy*, January, 1937) traces the development of the infraumbilical abdominal wall from a study of a series of embryos ranging from 1.4 to 40 millimetres in length. The infraumbilical portion of the abdominal wall, the genital tubercle, *symphysis pubis*, and muscular coat of the bladder, are formed from a well-defined band of mesoderm. This band has a twofold origin: (a) from the caudal margin of the embryonic shield—primary mesenchyme; (b) from processes of secondary mesoderm passing round the cloacal membrane from the hind end of the primitive streak. At an early stage the cloacal membrane is a relatively large area of contact of ectoderm and endoderm extending some distance along the allantoic diverticulum. This allantoic cloacal membrane is later obliterated by the mesoderm pressing in towards the mid-ventral line between the ectoderm and endoderm. Extroversion of the bladder is due to mesodermal deficiency, particularly of the processes of secondary mesoderm, following on which there is persistence of the primary extensive cloacal membrane with impaired development of the muscular coat of the bladder, of the *symphysis pubis*, and of the formation of the external genitals and infraumbilical portion of the anterior abdominal wall. The author also believes that epispadias is a similar mesodermal error in a minor form.

Cerebral Hemispheres of *Lacerta Viridis*.

AN investigation of the cerebral hemispheres of *Lacerta viridis* is described by F. GOLBY (*Journal of Anatomy*, April, 1937). The following information of myelinated tracts was obtained by the use of the Marchi degeneration technique. Primary olfactory fibres from the main olfactory bulb are for the most part relayed in the anterior olfactory nucleus before they pass to other fore-brain centres. The lateral olfactory tract consists predominantly of fibres from the accessory bulb, and many of them reach the amygdaloid nuclei

without an intermediate relay. There is no efferent projection tract from the antero-dorsal region of the cortex. There is some evidence for the presence of short efferent fibres from the cortex passing medially in the alveus system to the paraterminal body. The lateral forebrain bundle contains efferent fibres from the *corpus striatum* which can be traced into the subthalamic region. Many end here, probably connecting with the hypothalamus. A few can be followed through the tegmentum of the mid-brain. Stimulation and ablation experiments support the idea that the striatum, and probably other basal centres, function as coordinating mechanisms in the synthesis of motor behaviour patterns. They suggest that these centres participate in the control of muscular tone and that they can facilitate or inhibit motor reactions in lower levels of the nervous system. None of these experiments supports the theory that the antero-dorsal part of the cerebral cortex of reptiles is neopallial in nature.

Narial Margins in Man.

EDWIN H. JOHNSON (*Journal of Anatomy*, April, 1937) advances a theory to account for the variations of the narial margins in man. He suggests, from an examination of the developing premaxillary-maxillary region in white fetuses, that the duplication of the inferior narial margin, which is such a feature in certain skulls, is due to an incomplete passage of the incisor processes of the maxillae forwards, so that, although they still meet in the mid-line and form the anterior walls of the incisor sockets, they do not divorce the premaxillae from entering into the formation of the narial margin. Observations made on the ontogeny of the maxilla and premaxilla in man, and on their changing relationship in the primates, support the statement that the incisor processes of the maxillae are overgrowing the premaxillae in phylogeny. In the non-human primates the narial margins are completely premaxillary. In the prognathous types of man the margins are formed by the maxillae and premaxillae in degrees varying according to the amount of subnasal prognathism. This gives rise to duplication of the narial margin. In the orthognathous white the narial margins are completely maxillary and therefore single.

Cerebral Hemispheres of the Chimpanzee.

A. EARL WALKER AND JOHN F. FULTON (*Journal of Anatomy*, October, 1936) give a detailed description of the external configuration of the cerebral hemispheres of the chimpanzee. They found that the convolutional markings were, unlike those of most mammals, very variable. The homologies of these in man, anthropoids and monkeys is also discussed.

British Medical Association News.

SCIENTIFIC.

A MEETING of the New South Wales Branch of the British Medical Association was held on June 24, 1937, in the Robert H. Todd Assembly Hall, British Medical Association House, 135, Macquarie Street, Sydney, DR. LINDSAY A. DEX, the President, in the chair.

Vascular Diseases.

DR. GEORGE WILLCOCKS read a paper entitled "Vascular Disorders of the Limbs" (see page 121).

DR. G. F. S. DAVIES, in opening the discussion, thanked Dr. Willcocks for his paper. He said that a number of vascular diseases of the extremities were seldom encountered by the morbid anatomist. The chief problem was the diagnosis of *thrombo-angiitis obliterans* (Buerger's disease) and its differentiation from more common conditions which might imitate it. The material received for examination was mainly in the form of limbs or parts of limbs which had been amputated because of gangrene.

Ten recent cases of this sort were selected for discussion. They fell into three main groups. At one end of the scale were the senile and the diabetic patients, whose ages varied from sixty-five to seventy-seven years. At the other end of the scale were two patients, aged twenty-seven and twenty-eight years. One of these was thought to be suffering from Buerger's disease, the diagnosis in the other instance being doubtful, on account of the small amount of tissue received. In an intermediate group was a case which imitated Buerger's disease clinically, but in which this diagnosis was not supported by the histological evidence.

Dr. Davies showed lantern slides which illustrated some of the diagnostic features. A slide from a woman, aged seventy-seven years, illustrated Mönckeberg's sclerosis and atheroma. Here thrombosis followed a degenerative condition of the blood vessel wall. There was degeneration of the media with calcification and ossification. The intima was thick and hyaline, but the thickening was irregular. The fibres of the hyaline tissue tended to run in a circumferential direction. Lipoid was often seen in the intima and was in the form of cholesterol clefts or as lipoid histiocytes. Calcification might occur. Splitting of the intimal elastic lamina could be demonstrated. As a contrast, a slide from a case of Buerger's disease was shown next. Here a previous acute inflammation of the vessel wall was responsible for the original clotting of blood in the lumen. By the time the section material was obtained, the acute inflammation had subsided and the clot had become organized and recanalized. The adventitia was thick and fibrous, and bound the vessel to adjacent veins and nerves. The media contained larger *vasa vasorum* than a normal vessel. The intima showed little or no thickening, though slight splitting of the elastica might occur. The clot contained numerous small blood vessels of various sizes. There was intracellular blood pigment from the original thrombus.

Two slides from a diabetic patient exhibited advanced atheroma. In one there was no thrombosis, but changes characteristic of atheroma were seen. In a second slide there was a similar condition of the vessel wall, but with an organizing thrombus in the lumen. The next slide was from the clinically doubtful case previously referred to. Here there was an organizing thrombus, but the intima showed such gross irregular thickening and degeneration that the diagnosis of thrombosis following atheroma was made. The picture very closely imitated the previous section, which was from a diabetic. A final slide, made from a section of a left common carotid artery, showed how thrombosis followed by organization in a syphilitic artery could imitate Buerger's disease.

DR. H. C. RUTHERFORD DARLING, having congratulated Dr. Willcocks on his masterly exposition on the subject of

vascular diseases of the extremities, sought the President's permission to relate one case which would severely contest Dr. Willcocks's opinion that sympathectomy was of value in Raynaud's disease.

The patient was a Londoner by birth and had emigrated to Sydney in March, 1921. She was a shipping clerk of twenty-five years of age. Symptoms were first noticed during the six years prior to her leaving England, when for periods of three months each winter chilblains developed on the hands and feet, especially the former. During the first three winters in New South Wales no chilblains appeared, but for the last six years chilblains had been recurring with gradually increasing severity.

For the last three years the hands, forearms and legs, from the knee downwards, had always felt cold, and during the winter months the fingers had felt numb. During the four colder months the hands and feet had intermittently assumed a bluish colour and with this was associated a constant dull aching pain. This pain was not appreciably altered by rest or exercise, elevating or depressing the limb; and temporary relief for approximately half an hour might be obtained by immersing the limbs in warm (not hot) water and applying friction.

During the last three winters (1926, 1927 and 1928) superficial ulcers had developed on the fingers, thereby rendering the patient incapable of following her employment for recurring periods of approximately two months' duration. The most severe attack occurred in May, 1928, when "black scabs" appeared on the fingers of both hands.

During the above three years the patient had been constantly under medical treatment.

Dr. Darling went on to say that Sir Thomas Lewis had defined Raynaud's disease as a malady in which the fingers and toes became periodically pale or cyanotic, and in which, after many winters of repeated attacks, the terminal portions of the digits might be lost by a process of dry gangrene. The case quoted might therefore be regarded as a typical case of Raynaud's disease.

The left sympathetic cord had been sectioned on April 22, 1927, and the right cord on June 10, 1929. The immediate post-operative results were so excellent that Dr. Darling decided to report the case. Before submitting this infliction upon the medical profession, however, he had the wisdom to submit copies of the manuscript to Sir Thomas Lewis and Dr. Norman Royle.

The former pointed out that Raynaud's disease was primarily due to a local defect in the arterioles, whereby cold acted unduly upon the vessel wall, and this syndrome was certainly not due to any over-action of the sympathetic nervous system. He prophesied that the symptoms would recur in an unmitigated form within two years.

The subsequent history of this patient was most interesting. She had been exhibited at a clinical meeting of the Branch on October 25, 1934, and a clipping from THE MEDICAL JOURNAL OF AUSTRALIA of December 15, 1934, was shown.

The patient had been classed as suffering from "hysteria". This malady had been defined as a condition in which ideas controlled the body and produced morbid alterations in its functions. As both the pre-ganglionic and post-ganglionic fibres supplying the arterioles of the right upper limb had been divided, one should look upon the subsequent history as affording confirmation of Lewis's thesis that Raynaud's disease was primarily a local defect of the arterioles. Further, Horner's syndrome in this case could hardly be regarded as an hysterical manifestation. Dr. James Collier had issued frequent warnings that hysteria should never be used as a mere limbo in which to place ill-understood maladies, or those that evaded diagnosis and which did not present its characteristic features.

Like the proverbial bad penny this patient had again turned up in one of Dr. Darling's beds and was awaiting oophorectomy at his visit to the Prince Henry Hospital on February 1, 1935. Dr. Darling had pasted the clipping from THE MEDICAL JOURNAL OF AUSTRALIA of December 15, 1934, in the hospital notes and had advised her transferece to Broughton Hall.

But this was merely *au revoir*, for two months later what remained of the patient was listed at a clinical

meeting of the New South Wales Branch as a case of Raynaud's disease. At that meeting Dr. Darling had taken the opportunity of examining her abdomen, and had found a recent scar through which her last remaining ovary had been extracted. The psychiatrist's prognosis had been fulfilled.

Dr. Darling at this juncture drew attention to the delayed post-operative results of pre-ganglionic and post-ganglionic section. In the former the persistent vasoconstrictor discharges through the post-ganglionic fibres were temporarily abolished, whilst in the latter the smooth muscle of the blood vessels soon after denervation became hypersensitive to adrenaline circulating in the blood. The patient, who was last seen on June 17, 1937, still presented Raynaud's phenomena in her upper extremities, but this malady was now complicated by bilateral acrocyanosis, an affliction alleged to be curable by sympathectomy. Dr. Darling left it to the meeting to decide whether they would in future recommend sympathectomy as a treatment for obstinate cases of Raynaud's disease. He then went on to discuss the treatment of *thrombo-angiitis obliterans*. In the early stages of Buerger's disease the obliteration of the arteries was partly mechanical and partly spasmodic.

In instituting treatment it would be necessary to estimate the relative proportions of these two factors, for it should be obvious that heat, contrast baths, protein therapy, vaso-dilator drugs or operations on the sympathetic system *et cetera* could have no action in cases in which the obstruction was of purely mechanical origin.

The simplest method of estimating the amount of vasospasm in such cases was by recording the skin temperature before and after alteration of the environmental temperature—the so-called "blanket test". The patient lay naked in a warm room at a constant temperature of 25° C. (77° F.) for one hour, and readings were taken by a skin thermometer. The patient was then covered by a warmed light rubber sheet and wrapped in three blankets, and one hour later the skin temperature readings were again taken at the same points.

In those cases in which the obstruction was shown to be of mechanical origin, treatment should be directed towards enlarging the capillary bed, and took the form of hypertonic saline solution injections or vaso-ligation.

Dr. Darling was doubtful whether vaso-ligation delayed amputation in arteriosclerotic cases, but it was of definite benefit in *thrombo-angiitis obliterans*. Vaso-ligation was synonymous with ligation of the popliteal vein. His best case had been that of a barman, aged forty-eight years, who had had double vaso-ligation performed for severe rest pain in October, 1933. He resumed his employment and was well until 1936, when he accidentally stubbed his left big toe against a case of beer. A blood blister formed, which became infected and gave rise to necrosis of the terminal phalanx. In September, 1936, the nail was evulsed and the terminal phalanx enucleated, but the wound took many months to heal. When the patient was last seen, on June 24, although his left big and second toes were still cold and purplish in colour, he had no rest pain and could walk between two and five miles *per diem* without inducing discomfort.

Dr. HAROLD RITCHIE congratulated Dr. Willcocks on his presentation of an extremely difficult subject. He was pleased that Dr. Willcocks had spoken of the difficulty of distinguishing between the different disorders. Certain parts of medicine were overburdened by terminology, by authors wanting to give new names to old diseases. With regard to therapeutic measures for relief of vascular disorders of the extremities, Dr. Ritchie emphasized the dangers attendant upon the administration of hypertonic saline solution. It should be used with extreme caution, especially in elderly people. In these circumstances the hypertonicity should be low, not more than 2% or 3%, and the quantity should be small. It caused discomfort, flushing, headache, and sometimes what appeared to be true cardiac pain. With regard to acetylcholine, Dr. Ritchie referred to a conversation that he had had with Dr. Charles Kellaway, of Melbourne, who agreed with him that many proprietary preparations were inert, although it was now possible to procure acetylcholine

which was potent. He had been greatly interested by Dr. Davies's slides, which were informative and valuable, and showed that even the pathologist could not always be sure as to the diagnosis of some of these disorders. They were proof that there was a great deal to be learned about vascular diseases of the extremities. Medical practitioners owed it to their elderly patients to take great care of their peripheral circulation and to prevail on the patients to do the same. Further, in the examination of patients an attempt should always be made to estimate the integrity of the peripheral circulation.

Dr. Willcocks, in reply, apologized for having presented a paper which took so long to deliver, but said that the subject was a vast one. He thanked Dr. Davies for the slides that he had shown, and Dr. Ritchie for what he had said, particularly in regard to the care necessary in giving intravenous injections of hypertonic saline solution to elderly patients. It was quite true that such injections could give rise to serious symptoms, and incidentally the administration of large injections was a clumsy procedure. It was difficult for the medical practitioner to know what to do for these patients, but the case quoted showed what conservative treatment would do, although such treatment took a long time, and patients objected to it. Dr. Willcocks stated that he did not advocate sympathectomy or stripping of the vessels; he had had experience of only one case, and the result had been moderately good. He did not consider Raynaud's disease a common condition in this country, and he would hesitate for a long time before advocating sympathectomy as a means of treating it. He was very interested in the letter which Dr. Darling had received from Sir Thomas Lewis, giving the probable results of sympathectomy in the case that Dr. Darling had quoted. Much observation was necessary before any such operation was performed on a patient suffering from Raynaud's disease.

A MEETING of the Victorian Branch of the British Medical Association was held at the Royal Melbourne Hospital, Melbourne, on Wednesday, May 19, 1937. The meeting took the form of a number of clinical demonstrations by members of the honorary staff. Parts of this report appeared in the issues of July 10 and 17, 1937.

Conservative Treatment of Thyreotoxicosis.

Dr. GEOFFREY A. PENINGTON presented a series of patients illustrating the conservative treatment of thyreotoxicosis. He said that he wished to show that in carefully selected cases it was possible to obtain results that were as good as might be expected from surgical intervention. He pointed out that there were many patients with this disease who presented themselves for examination at a stage when it was difficult to be sure on clinical grounds whether they were suffering from thyreotoxicosis, neurasthenia, the "effort syndrome", chronic sepsis or the exhausting effects of the strain and stress of modern life, especially when the last-mentioned was aggravated by financial and domestic worry. The factors which caused the difficulty in diagnosis were in themselves a common cause of the onset of toxic goitre in those persons who were predisposed to the disease, and thus increased the difficulty. During the period of observation of these patients it was possible to obtain a good estimate of the likelihood of response to conservative measures; and in those instances in which the disease was frankly manifest there was rarely any need for an immediate decision regarding the desirability of performing surgical operation. Dr. Penington said that persons suffering from primary or secondary thyreotoxicosis of short duration, those suffering from the *forme fruste*, patients who refused to have an operation for personal reasons, and those who would be unable to have the services of a first-class "team", were the types that should be given a thorough trial with medical treatment. He thought that in every instance it was necessary to inquire thoroughly into the economic possibility of a prolonged period of rest, and to study the various psychological factors at work, and above all not to neglect the great importance of the environment to which the patient

had to return during and after recovery. The time saved as a result of thyroidectomy might compel the physician to advise surgery in some instances. It was suggested that a good response to rest, improvement in the general condition of the patient, and a gain in weight should encourage the trial of medical treatment, which could be persisted in if progress was steadily maintained, if the basal metabolic rate was falling, and if the degree of invalidity was slight during this period. Dr. Penington also stressed the point that the treatment of a patient with thyrotoxicosis should be directed to the individual and not to the thyroid gland, which was only one part of the disease entity. He said that if the gland was made the sole object of the attack, although the basal metabolic rate would fall, the patient would not be cured. Medical treatment consisted in the judicious use of regulated rest, both physical and mental, forced feeding, the removal of sepsis, and the careful use of sedatives and other medicaments. Radiography was also a great help in many cases. The patient should be carefully watched for at least two years to ensure that there was no relapse.

The first patient shown by Dr. Penington was a married woman, thirty-one years of age. She had been seen on October 29, 1936, and complained of having felt the heat during the preceding two years, and of recurrent attacks of epigastric pain for five months. The pain had no relation to meals, was aching in character and was unrelieved by home remedies. She had lost eight pounds in weight during the latter period and had noticed palpitation, nervousness and shortness of breath; she had had a great deal of worry, domestic and financial. Dr. Penington said that when the patient was examined she had had a warm, moist skin, was very thin, and the mucous membranes were pale. No eye signs were present, but there was a pronounced tremor of the hands; the systolic blood pressure was 103 and the diastolic pressure 80 millimetres of mercury; and the pulse rate was 120 per minute. The thyroid gland was diffusely enlarged and soft, with a systolic bruit to be heard over it. The tonsils were septic and a mild degree of secondary anaemia was present; cholecystography revealed no abnormality; the basal metabolic rate was +42%. Dr. Penington remarked that owing to her financial condition the patient could not rest adequately at first, and, despite sedatives, the basal metabolic rate was +66% in December. Assistance had been arranged by the hospital almoner, and in February the basal metabolic rate with the patient at rest in hospital was found to be 0%, and all signs of toxicity had disappeared; no iodine had been given at any stage of the treatment. During February and March the patient was resting away from her home and two children; she gained two stone in weight and remained well and resumed her work again. The basal metabolic rate was +6% on May 7. Recovery seemed to be complete, and the pain had entirely disappeared during the treatment.

Another patient shown by Dr. Penington was a married woman, twenty-six years of age. She furnished a typical example of exophthalmic goitre and had come under Dr. Penington's observation on December 7, 1935, with the history that she had been in indifferent health during pregnancy, which had terminated in July, and that one month later she had developed tremor, palpitation, dyspnoea on exertion, and nervousness, irritability and sweating. She had lost weight, and although she had been given Lugol's solution for one month in October, her condition had become worse. Exophthalmos, a wide palpebral fissure and lid-lag were extreme; the skin was warm and moist; tremor, nervousness and restlessness were evident. The systolic blood pressure was 120 and the diastolic pressure 70 millimetres of mercury, and the pulse rate was 140 per minute. The thyroid gland was uniformly enlarged and firm; a double bruit was audible over it; the basal metabolic rate was +43% on December 10. Dr. Penington said that it was thought that it would be necessary for her to submit to operation, and with this in view she had been admitted to the medical ward; but after five days in bed and with only fifteen minims of Lugol's solution daily her condition had greatly improved. The pulse rate while she was asleep was sixty-five per

minute, the patient had gained weight, and there was a definite loss of nervousness. In view of this improvement it was decided to try conservative measures. "Luminal" was given in a dose of half a grain three times a day and X ray therapy was commenced. After one month in hospital the patient was returned to the out-patient department, and during the next two weeks she gained seven pounds in weight; on January 16, 1936, the basal metabolic rate was +36%. The patient had returned to her home and four children with instructions to rest, and these were faithfully carried out with the assistance of relatives; only one course of X ray therapy had been given, but she had continued to take "Luminal" and had reported well in July. In April, 1937, the basal metabolic rate was -15%; she had gained in all one and a half stone in weight and was very well, although there had been no decrease in the exophthalmos. It was proposed to give her eserine in an attempt to improve this condition. Dr. Penington said that the result in the treatment of this patient was quite as good as could be expected from expert surgical treatment; but he mentioned the importance of continued observation and the possibility of the patient's having to undergo further X ray treatment in the future.

Another patient shown by Dr. Penington was a man, fifty-eight years of age, who had been under treatment since March 4, when he had complained of loss of weight (over one stone) for five months, a dislike of warm weather, sweating at night, and a dry cough for three months; there had been no other symptoms. The patient was florid, had slight exophthalmos, a widened palpebral fissure, lid-lag, and a fine tremor of the hands; the thyroid gland was palpable, but was not obviously enlarged; the pulse rate was 100 per minute; the systolic blood pressure was 160 and the diastolic pressure 100 millimetres of mercury. After radiographic examination of the chest it was reported that there was no retrosternal dullness; an old fibrous lesion was present in the third right interspace, but there was no evidence of any active trouble; the left vocal cord moved sluggishly, and the basal metabolic rate on March 5 was +40%. On March 8 auricular fibrillation was present, with a heart rate of 144 per minute; but when the patient was admitted to hospital on March 10 the rhythm was normal and the heart rate had dropped to 88 per minute; the systolic blood pressure was 135 and the diastolic pressure 85 millimetres of mercury. A confirmatory test showed that the basal metabolic rate had risen to +60%. The pulse rate fell rapidly to 72 per minute on "Luminal" treatment with Lugol's solution in a dose of ten minims three times a day. As there was no thyroid enlargement and apparent good health had been so rapidly restored, it was decided to try medical treatment. The patient had rested in the country for six weeks while taking "Luminal" and quinine hydrobromide, and by May 6 he had gained one and a half stone in weight; the pulse rate was 82 per minute, the systolic blood pressure was 152 and the diastolic pressure 80 millimetres of mercury. The patient complained of precordial pain and tightness on exertion, and although this became less when the dose of quinine hydrobromide was increased to ten grains three times a day, it was viewed with some disquiet, as two brothers had died suddenly at about the same age. An electrocardiogram had revealed no abnormality, but Dr. Penington considered that it was necessary to reconsider the advisability of surgery in view of this development. He had decided that it was justifiable to persevere with X ray therapy, which had been commenced in May. He said that unless there was some further improvement during the next few weeks he would again review the position.

Dr. Penington remarked that in presenting another patient he was demonstrating what he considered to be a poor result; but he had been forced into giving the patient medical treatment because of her refusal to have the second stage of the operation performed, and the precipitating cause of the condition was still irremovable. This patient, a married woman, forty-four years of age, had reported on April 27, 1933, that she had suffered for two years from headaches, nervousness, palpitation, dyspnoea on exertion, loss of appetite and weight, and severe skin

irritation all over the body. She had had diarrhoea at intervals during that time; her eyes had been prominent for eighteen months and tremor had been noticed for two months. There were also typical signs of exophthalmic goitre with prominent nervous manifestations and slight enlargement of the gland. The pulse rate was 140 beats per minute; the systolic blood pressure was 165 and the diastolic pressure 75 millimetres of mercury. The basal metabolic rate was +67%. The patient had been admitted to the hospital on May 3, 1933, and right hemi-thyroidectomy had been performed on May 22, after the usual pre-operative treatment, and she had left the hospital greatly improved, but still toxic. In August the basal metabolic rate was +48%, and she was urged to have the second stage of the operation performed; but she refused for personal and domestic reasons. She was then given small doses of Lugol's solution (five minims three times a day) for two periods of two weeks, and quinine hydrobromide and "Luminal" continually until November 23; the basal metabolic rate was then found to be -17%. Dr. Penington said that at that stage there still appeared to be some toxicity, and the skin irritation was also troublesome. The patient had not reappeared at the hospital until May 27, 1935, when she complained that the original symptoms had returned. The pulse rate was 104 per minute; the systolic blood pressure was 180 and the diastolic pressure 90 millimetres of mercury. The patient had again refused to have the second operation, so she was given "Luminal", bromides and quinine, and without any further investigation of the basal metabolic rate X ray therapy was commenced in August. Dr. Penington said that, though there was some improvement in her condition, she still appeared to be toxic when on November 25, 1935, the basal metabolic rate was found to be -33%. On February 24, 1936, the basal metabolic rate was estimated again, the result being +12%. The patient did not appear again at the clinic until April 29, 1937, when the same symptoms were present, and Dr. Penington had made her the subject for a clinical demonstration of the classical signs and symptoms of thyrotoxicosis for the benefit of his students. He said that it was anomalous that she had gained in weight, and this was explained when it was found that on May 7 the basal metabolic rate was -10%. The patient was having large doses of sedatives and an hypnotic at night, with resulting diminution of her symptoms and signs. Dr. Penington stressed the point that there was still a great deal of worry and sickness in the home, the effect of which the patient was quite unable to avoid, and that the onset of the condition had been coincident with the occurrence of these difficulties. She had been subjected to this stress continually, and he thought that this was one reason for the poor result. This case demonstrated that treatment of the "lump in the neck" would not result in a cure, and that the nervous *Asiatic* and persistent symptoms were sometimes unaffected by controlling the metabolism. The patient provided another argument in favour of the one-stage operation.

Myotonia Congenita (Thomsen's Disease).

Dr. E. GRAEME ROBERTSON showed two patients together—Mrs. E. and her son, J.E. The boy J.E., nineteen years of age, stated that after manual work his hands closed up and his arms became powerless, a symptom which had been troublesome for as long as he could remember. He also said that his hands were prone to become stiff; if he was riding his cycle and his hands got cold he would be unable to open them. He said that his hands tended to take the shape of the object that he was holding. After doing anything requiring a tight grip, he at length could hold the object no longer and might be unable to open his hands for one or two hours. He knew the condition was commencing when his forearms seemed to lose their strength, and he felt a painful tenseness in the muscles in the anterior aspect of the wrist and lower part of the forearm. The difficulty was worse in cold weather, when it developed more quickly and his fingers spontaneously curled up. The patient said that if he ran for a tram at the terminus and then slowed down, but had to break into a run to catch the tram, he could feel the muscles

stiffen at the back of his knees; this might last for about two minutes and force him to slow down to a walk, then the stiffness would gradually disappear and he could break into a run again. The patient never had any difficulty in breaking into a run or in starting to walk.

After examination it was found that there was difficulty in relaxing a continued powerful grip; tonic flexion of the wrist and tight contraction of the flexors of the fingers developed, which could be relaxed only with difficulty, and then very slowly. Otherwise neurological and general examination revealed no abnormality. The muscular power was excellent; no contraction of muscles was induced by direct percussion. Dr. Robertson remarked that when the electrical reactions of biceps, forearm muscles, calf and peroneal muscles were tested, a brisk normal contraction was found in all muscles except those supplied by the left peroneal nerve, where a pronounced myotonic reaction was detected. It was thus interesting to note that the typical mode of reaction was absent from the muscles most often affected and was present in muscles but rarely affected. Had the myotonia been induced by grasping, no doubt the reaction would have been obtained in the forearm muscles.

The patient's family history showed that his maternal grandmother had suffered from the same condition. Her hands were known to have "cramped up", and she had also suffered from "chalky gout". She died of pneumonia at the age of seventy-four years. The boy's mother also suffered from the same condition; she stated that she had had the symptoms as long as she could remember, and that they were particularly related to the cold. When she was washing clothes she had to dry her hands before hanging out the clothes, otherwise she could not hold the pegs. She wore gloves to keep her hands warm and supple. The cramp was also induced by lifting weights, and was demonstrated even better than in her son by continued grip; she developed cramps in her legs when she stretched them, but otherwise noticed nothing unusual in her lower limbs. The mother had one older and one younger sister, who had no trace of the condition. She had four sons, the eldest of whom was twenty-seven years of age and who had had the condition since infancy; it interfered with his work, but had not been so bad lately, as he had been working indoors. Another son, twenty-five years of age, was normal. The third son, the patient described by Dr. Robertson, suffered from the condition, and the youngest, sixteen years of age, was perfectly healthy.

Dr. Robertson said that in regard to the treatment of this condition Wolf had shown that quinine by intravenous injection or by mouth removed the myotonia. Foster, Kennedy and Wolf concluded that prostigmin was effective in myasthenia by virtue of its catalytic influence on acetylcholine, while quinine was effective through its inhibition of acetylcholine, both acting at the myoneural junction. The boy had received great benefit from treatment with quinine hydrochloride, given by mouth three times a day in doses of five grains.

Gigantism.

Another patient shown by Dr. Robertson was a man, twenty-six years of age, who had been admitted to hospital on August 12, 1936, complaining of continuous headaches. In 1931 he had sustained fractures of the spine and skull in the New Zealand earthquake. On May 7, 1936, he had struck himself on the vertex with a sixteen-pound hammer and was unconscious for half an hour. The patient had been able to walk to hospital, but when admitted he was kept in bed for about seven weeks, complaining of almost continuous headache, starting over the forehead and passing over the vertex to the occiput. The pain was relieved by recumbency and increased by the assumption of the erect posture. A skiagram of the patient's skull showed a smooth, linear defect in the left frontal and temporal regions, possibly of an old fracture. The patient was a typical giant, six feet seven inches in height. There were no abnormal neurological physical signs, and the visual acuity of the right eye was $\frac{1}{20}$. The acuity of the left eye was $\frac{1}{20}$. The visual fields, including the colour fields, were full; the fundi were normal. A skiagram of his skull revealed a slight but definite enlarge-

ment of the *sella turcica*, and the cerebro-spinal fluid was under an initial pressure of 140 millimetres and was clear and colourless. No cells were seen in three cubic millimetres of fluid; the total protein was 0.030%, and there was no increase in globulin. The colloidal gold curve was 0000000000. The Wassermann test gave no reaction with either the blood serum or the cerebro-spinal fluid. The basal metabolic rate was -32% and the glucose tolerance curve was normal.

Dr. Robertson said that the headache seemed to be disproportionately severe for the degree of enlargement of the pituitary gland, and its onset was associated with the head injury. Continued rest in bed, however, had failed to produce relief when the patient assumed the erect posture. Eventually deep X ray treatment was given in two full courses to the pituitary gland, but the patient still complained of the headache. The definition of the posterior clinoid process had improved.

Dr. Robertson showed the patient's family tree. The grandfather had been six feet nine inches in height, and the father six feet two inches. The patient was one of seven brothers, all but one of whom were over six feet two inches in height.

Pseudohypertrophic Muscular Dystrophy.

Another patient shown by Dr. Robertson was a boy, aged five years and seven months. The patient had not walked until he was nineteen months old, and then he had always walked peculiarly; although he had looked strong he was easily knocked over. His mother had noticed that his feet were peculiar; "the instep was enlarged and the sinews contracted". Examination showed him to be a fine, bright boy, who looked like a miniature Hercules. He stood with a lordosis and had very large calf muscles, which had the hard, rubbery feeling characteristic of pseudohypertrophic muscular dystrophy; he walked with a waddling gait. His smile was transverse, lacking the upward movement of the corners of the mouth. When the patient wished to rise from the supine position he rolled on his front, then he got on to all four limbs and "climbed up his legs". A more detailed examination revealed weakness of the flexors of the neck, all the shoulder girdle muscles and muscles of the arms, trapezi and *latissimus dorsi* muscles. The flexors of the spine and hip were also very weak; the hypertrophied *quadriceps femoris* and calf muscles were of fair power, and the latter were showing contracture; the dorsiflexors of the feet were weak. Bilateral *talipes equinovarus* was present; the deep reflexes were all absent, except the ankle jerks, which were feeble. The patient had small external genitalia and an undescended testicle. He had been treated with glycine (ten grains a day), which had not stayed the course of the disease. Although six months prior to the meeting he had been able to rise from the lying position unaided, he had become unable to do so; "his heels did not reach the ground as they used to", and the shoulder girdle muscles were definitely much weaker. The family history of the patient showed that the patient's grandparents had both been normal, but although his mother, thirty years of age, believed herself to be well, she had very obvious bilateral ptosis and lordosis; on inquiry she stated that she had never been able to sit up from a lying position without the assistance of her arms. The mother said that for seven years she had suffered from shortness of breath and palpitation on exertion, and for six months had aching limbs and dull pains in the small of her back. On examination, weakness of the *orbicularis oris* and *levator palpebrae superioris* muscles was obvious. The infraspinatus and calf muscles were increased in bulk and had the characteristic rubbery feeling of the disease. Dr. Robertson thought that it was interesting that the patient was unaware of having the family disease, and that she presented a combination of two different types of myopathy, the involvement of either type being minimal. She had two sisters, one of whom had had two sons and one daughter; the two sons had both died at the age of twenty-one, and had appeared to be "very helpless". The daughter, who was thirty years of age, walked with "stiff hips" and

was gradually getting worse. Her other sister's son had died at the age of twelve years from a similar disease. The patient described was an only child.

Narcolepsy.

Dr. Robertson also showed a male patient, twenty-five years of age, who had suffered from headaches at varying intervals for twelve years. They always commenced in the morning and lasted for several hours or until sleep supervened; they started behind the left frontal region and passed to the right side behind the eye; when they were very severe the patient vomited and became prostrated, having to lie down in a darkened room. His vision frequently became blurred for several hours during the attacks, and on one occasion during a period of headache he was unable to judge whether a horse and cart were coming towards him on one side of the road or retreating on the other, and he was forced to dismount from his bicycle. The headaches had ceased after this attack. The patient said that at intervals for seven years, for no apparent reason, distant objects had appeared doubled, whether the left eye was open or shut. In April, 1936, the patient had noticed, while climbing a hill on his way to work, that he lost his sight, felt giddy and nauseated, and, unless he stood still, toppled over and lost consciousness. One month later he found that when he became excited or started laughing his knees would give way under him and he would fall down. He said that he connected that type of attack with laughing or talking too much, and sometimes with exertion, so he had avoided laughing as much as possible "because a man doesn't want to make a fool of himself". He described one such attack when he had been laughing and joking with his family and suddenly "the feeling came all over him" and his legs had given way; he had lain on the floor, able to hear what was said to him, but unable to move a muscle; eventually he had been able to roll his head to the left side and then to lift it and to stand erect. He also remembered that while playing cricket an "easy catch" had been coming directly towards him, but he had trembled all over and the ball had passed through his hands. He said that people told him that during some attacks he had made silly statements, disconnected with the previous talk, yet he himself had not been aware of saying anything. He had found himself wobbling when riding his bicycle, and even riding on the footpath. In August the patient had commenced to suffer from attacks of overpowering drowsiness which had previously been quite foreign to him; this usually occurred at meal times, when he was reading a book, or while "having a rest", leaning against a tree after loading a truck. The attacks lasted for five or ten minutes and made him the laughing stock of his fellow relief workers. The patient drank about five pints of water or tea a day and passed large quantities of urine. Dr. Robertson said that a complete neurological and general examination had revealed no abnormalities, and when the patient had complained of diplopia no reason could be found for it. The patient said that diplopia was present when the right eye alone was open, but not the left. During the examination attempts had been made to provoke laughter, with little result except to increase the patient's seriousness. After the examination, however, he had been talking animatedly to his neighbour, saying that he was never going back on relief work, when suddenly he had ceased speaking, his eyes had screwed up and there was a rapid tremor of his jaw; all his limbs had been flaccid, with poor deep reflexes and flexor plantar responses. Before other examinations could be carried out, however, he recovered and realized that "it had come over him". The examination immediately afterwards had yielded perfectly normal findings.

Dr. Robertson said that on account of the many interesting and unusual features, the patient's condition had been somewhat fully investigated, on the chance that some abnormality in the region of the floor of the third ventricle would be demonstrable; all had, however, been normal. The cerebro-spinal fluid had been clear and colourless, under an initial pressure of 50 millimetres;

there were three large mononuclear cells per cubic millimetre of blood; the total protein was 0.04%; there was no increase in the hæmoglobin, and the colloidal gold curve was 0000000000. The Wassermann test gave no reaction; the blood urea was estimated at 24 milligrammes per centum; the glucose tolerance curve was normal; a skiagram of the skull was normal; there was a calcified pineal gland in the mid-line, and encephalography showed the lateral and third ventricles to be normal. The patient's grandparents were both normal; his father, who was fifty-six years of age, and his mother, who was forty-seven years of age, were both alive and healthy. The patient's mother's sister's child had had epilepsy and died at the age of sixteen years of heart failure. The patient had seven brothers and one sister. He was the eldest of the family, and all the rest of the family were healthy except his youngest brother, who suffered from epilepsy. The boy was seven years of age, and the fits had commenced two years earlier, after an attack of colitis, and had been of all degrees of severity: rare generalized tonic convulsions with slight clonic movement and extremely frequent minor attacks (as often as thirty a day), in which his head might nod at the table, or he might fall down at play and immediately pick himself up again. Treatment had had little influence in controlling the minor attacks, and mental deterioration had been rapid. Dr. Robertson said that there had been much discussion as to the relationship, or absence of relationship, between epilepsy and narcolepsy. The patient had presented symptoms suggesting migraine, cataplexy, narcolepsy and epilepsy. Dr. Robertson said that the striking superficial resemblance to the brother's attacks in their periodicity, abrupt onset with diminution or loss of consciousness, brief duration and rapid recovery did not escape the family. Yet it was possible that the incidence of narcolepsy in one member and of epilepsy in another was a coincidence. The patient had been relieved of the attacks of irresistible sleep by benzidine sulphate (20 milligrammes night and morning), which had also relieved, but had not cured, the cataplectic attacks. Dr. Robertson thought that it was of interest to note that a pharmacist supplied another patient suffering from the same disease with benzidine sulphate, in its proper dosage, instead of the correct drug. The error had fortunately been quickly discovered, but the patient was certain that he obtained almost as much benefit from the benzidine as from the benzidine. There was no chemical similarity between the two drugs.

Hypochromic Anæmia following Gastro-Enterostomy.

DR. L. E. HURLEY showed a female patient, forty-five years of age, who had been admitted to the Royal Melbourne Hospital on April 16, 1937. Nine years earlier she had been operated on for a ruptured gastric ulcer. The ulcer had been "infolded" and a gastro-enterostomy done. Four years before her admission to hospital the patient had had pneumonia. She stated that she had always been pale; but particularly pale in the four years after operation, during which she had felt tired and lacking in energy. In the past twelve months she had noticed a "pins-and-needles" feeling in the legs. The patient said that three months previous to the meeting a small ulcer had developed on the inner aspect of the lower third of the left leg; there had been some slight difficulty in swallowing, also some fullness and feeling of distension in the epigastric region after meals. On examination the patient looked very pale, with a slight icteric tinge; the tongue was clear and atrophic; there were cracks at the angles of the mouth, and several of the finger-nails showed spoon-shaped depressions; the edge of the liver was hard and could be felt three fingers' breadth below the costal margin. The spleen was not palpable.

On examination of the blood the red cells numbered 5,230,000 and the leucocytes 6,800 per cubic millimetre; the colour index was 0.49 and the hæmoglobin had been estimated at 42%. A film showed the presence of anisocytosis, definite hypochromia, and that the reticulocytes were less than 1%; there was neutropenia with a relative increase in the lymphocytes. The blood fragility test gave

a normal result, and the Wassermann test yielded no reaction. The Van den Bergh test gave a delayed positive reaction with 2.5 units of bilirubin; the result of the benzoic acid test of liver excretion was 1.4 grammes in four hours (normal three or more); the basal metabolic rate was -10%; no occult blood was found in the faeces, and a test meal showed an absence of hydrochloric acid.

Dr. Hurley said that, although the patient had been intensively treated with iron and hydrochloric acid ever since admission, there had been no material improvement, the hæmoglobin value on May 13, 1937, being still only 46%; other features of the blood examination had remained essentially the same as at the time of admission. Skiagraphic examination had shown some scarring of the duodenal cap, and most of the meals had left the stomach through the gastro-enterostomy opening. Dr. Hurley remarked that the patient had been shown as suffering from hypochromic anæmia, probably following a gastro-enterostomy; and he pointed out that in such cases response to the usual treatment with iron and hydrochloric acid was sometimes unsatisfactory. In the patient shown, deficient liver function might be partly responsible for the failure to respond to treatment. Further treatment with iron and hydrochloric acid in addition to liver would be tried, and if these failed to produce improvement a blood transfusion would be given. Expression of opinion was invited as to the possible benefit to be gained by "undoing" the gastro-enterostomy. The subject of anæmia following operation on the stomach and intestines was also discussed.

Cerebro-Spinal Syphilis.

Another patient shown by Dr. Hurley was a female, forty-five years of age, who had been admitted to hospital on March 22, 1937. The patient had had six healthy children and there had been no miscarriages. Two years earlier, over a period of six months, she had had a number of attacks of transient right-sided hemiplegia, and eight months before her admission to hospital she had noticed numbness and lack of feeling in the right leg; four months later she had complained of difficulty of micturition, which had become progressively worse in the last four weeks, when she had noticed a "tumour" in the lower part of the abdomen. Six weeks before her admission to hospital the patient had started to drag the left leg, which had become progressively weaker and stiffer, and for twelve months she had had diplopia.

Dr. Hurley said that on examination there had been a definite spastic weakness of the left lower limb, with the usual signs of a pyramidal lesion; on the right side there was loss of pain and of temperature sensation up to the level of the costal margin. Both plantar reflexes were extensor in type. The patient had slight loss of power in the right lower limb, and diplopia occurred on looking in all directions except to the left and downwards. The bladder was distended and could be seen and felt extending up to the level of the umbilicus. Examination of the eye grounds showed a patch of chorioiditis above the left optic disk. In the skiagram of the skull could be seen evidence of some bony thickening, and a retrosternal goitre was apparent in the chest films. Four red cells, some lymphocytes and one macrocyte were counted in each cubic millimetre of cerebro-spinal fluid. The total protein was estimated at 40 milligrammes per 100 cubic centimetres; the luetic curve was obtained with the colloidal gold test, and the cerebro-spinal fluid as well as the blood serum yielded a strongly positive Wassermann reaction. There was a moderate degree of hypochromic anæmia, the hæmoglobin value being 68%. Treatment was instituted by the administration of potassium iodide in a dose of 30 grains three times a day, by inunction of one drachm of blue ointment every day, and by regular catheterization at an interval of twelve hours. On April 9, 1937, treatment by means of "Novarsenobillon" was started with an injection of 0.3 gramme.

Dr. Hurley said that rapid improvement had occurred in the condition of the patient, and at the time of the meeting bladder function had been completely regained; the signs of pyramidal involvement had completely disappeared, but the sensory changes were unaffected. He

discussed the prognosis and treatment of cerebro-spinal syphilis and pointed out that the probability of improvement depended very largely on the nature of the underlying pathological process. In those cases in which the main phenomena developed very rapidly and there was probably some thrombosis, response to treatment was not as a rule very satisfactory. Long-standing lesions, in which fibrosis and secondary nerve degeneration had occurred, were also not very favourable. On the other hand, meningeal lesions and granulomatous lesions of a few weeks' or a few months' duration often gave a very good response to treatment.

Mitral Stenosis with Heart Failure.

Dr. Hurley also showed a male patient, thirty-one years of age, who had been admitted to the hospital on April 24, 1937. He had been well until about ten months before admission, when he had complained of shortness of breath on exertion. Shortly afterwards he had had "wheezing" attacks at night and had noticed that his heart was beating rapidly. He stated that at times his pulse was quite slow, and at others, particularly after any excitement, it became very rapid. He had remained very short of breath, even on slight exertion, and three months before admission had had to rest completely. At that time Dr. Hurley said that the patient had oedema of the legs and some "fluid" in the abdomen, but he had not lost any weight.

Dr. Hurley said that on examination the patient had appeared a little pale and was obviously short of breath; the veins of the neck were distended and pulsating; there was a diffuse swelling in the neck above the clavicle, but there was no definitely outlined mass. The cardiac apex was in the fifth intercostal space and four and a half inches from the mid-line. There was one finger's breadth of right cardiac dullness. Auricular fibrillation was present and had been confirmed by electrocardiographic examination; there was moderate congestion of the lung bases and also some pitting oedema of the lower limbs and sacral region. At the cardiac apex there was a systolic and also a rough diastolic murmur; the liver was palpable four fingers' breadth below the costal margin, and the edge of the spleen could be felt. Dr. Hurley stated that there was a moderate degree of hypochromic anaemia, the haemoglobin value being 68%, and the differential count of the white cells showed relative lymphopenia; the blood reaction to the Wassermann test was "negative". After a skiagraphic examination of the chest it was reported that there was a general enlargement of the heart with some pulmonary congestion. The basal metabolic rate had been +46% on admission, and six weeks later it had dropped to +23%. The patient was put under treatment consisting of rest, digitals and diuretics, with considerable improvement. The pulse rate had steadied down from 124 beats per minute on admission to between 70 and 80 per minute, and the oedema of the legs and the pulmonary congestion had disappeared. The diffuse swelling above the clavicle was also much less evident.

Dr. Hurley discussed the differential diagnosis of heart failure with auricular fibrillation. He said that in some cases it was very difficult to determine whether there was any underlying thyrotoxic basis. In the presence of heart failure the basal metabolic rate was sometimes elevated, when there was no thyrotoxicosis present.

Labyrinthitis, Meningitis and Facial Nerve Repair.

Dr. T. G. MILLAR showed a male patient, who had been admitted to hospital in July, 1935, suffering from severe vertigo and vomiting, and a pain in the left ear, which had been discharging for some years, the clinical picture being typical of acute septic labyrinthitis. Examination revealed nystagmus to both sides, and the patient lay on his side and was disinclined to move; no neck stiffness was present. The day after admission the vertigo was less and the patient was able to eat his dinner; but on the following day he complained of a headache, and it was noted that his neck was stiff; the Kernig test gave a positive result. A lumbar puncture returned turbid fluid, which showed 4,000 cells per cubic millimetre, most

of which were polymorphonuclear leucocytes, and haemolytic streptococci had been grown on culture. An operation was advised, and labyrinthine drainage according to the method of Neumann was performed. Pus escaped from the posterior semi-circular canal; profuse cerebro-spinal fluid was obtained from the internal auditory canal; recovery was rapid and no further lumbar punctures were required. Dr. Millar said that facial paralysis was present immediately after the operation, but this was anticipated, owing to the great technical difficulty during the labyrinth operation. Electrical tests carried out during the four weeks after operation indicated a poor chance of recovery of function. Dr. Millar decided to attempt a facial nerve graft according to the method of Ballance-Duel, and Dr. Hughes-Jones prepared a section of nerve in the thigh. In September the damaged nerve was dissected out and was found to be crushed in the region anterior to the lateral semi-circular canal; it was found possible to approximate the cut ends of the nerve after clearing away adjacent scar tissue, and the repair was covered by a piece of platinum foil, over which bismuth-iodoform-paraffin paste packing was inserted. Dr. Millar stated that after dressings had been performed in the usual fashion for a radical mastoidectomy, careful control was carried out by the physiotherapy department, and the first voluntary movement was obtained in the eighth month after the repair. He said that from then onwards there was progressive recovery of voluntary movement, but emotional movements were still defective.

Left Temporo-Sphenoidal Abscess.

Dr. Millar also showed a female patient who had had a chronic discharge from the left ear for years. Early in January, 1937, she suffered from tonsillitis followed by quinsy, and the left ear flared up and discharged profusely. When seen on January 20, 1937, the patient was drowsy and had vomited several times in the preceding twenty-four hours. On examination the patient was semi-conscious, with slow cerebration and suggestive aphasia. The right knee jerk response was very accentuated and the right extensor plantar reflex appeared to be extensor. There was a foul pulsating discharge from granulations obscuring the tympanic membrane; the temperature was normal and the pulse rate fifty per minute. On January 21, 1937, operation was performed and purulent mastoiditis was found to be present. The cells were thoroughly cleaned out, and the *dura mater* of the middle fossa was widely exposed. An area of *dura* in the tegmen region was found to be covered with granulations; the *dura* was not pulsating. A tiny opening was made in this area, and Heath's forceps were carefully inserted into the left temporo-sphenoidal lobe vertically; copious clear fluid followed by offensive pus was evacuated, and gentle palpation with the forceps suggested the presence of a large encapsulated cavity. A tube was inserted and the wound kept widely open with a bismuth-iodoform-paraffin paste pack. The patient was very well for five days after the operation, but on the fifth night she became drowsy again. She remained semi-conscious for four days and passed urine into the bed, but swallowed fluids. Weakness of the right arm and copious drainage of pus were present. Two days later the patient regained consciousness and recovered power in her right arm. The tube was removed from the wound on February 10, 1937, and Heath's forceps were passed along the track daily; there was less pus each day. In due course the wound was resutured under local anaesthesia and convalescence was uneventful.

Pansinusitis.

Dr. Millar also showed a female patient who had been admitted to hospital on November 24, 1936, with a history of nasal obstruction, with occasional colds and headaches over the frontal region. She had had a tender swelling over the forehead for two months, and some nasal discharge. On examination the tonsils were found to be enlarged and congested, and there was muco-pus on the fauces and pharynx; the mucosa of the nares was red and gelatinous, and polyps were present in the right middle meatus. On the forehead the swelling extended to both eyelids, the root of the nose and the upper part of the

face, and the affected area was slightly tender. Pus was obtained on both sides by means of antral wash-outs. In the skiagram the antra appeared dull and the frontal sinuses were trilobed, with poor transradiancy. Five days later the swelling over the forehead was fluctuant, and on the following day the right frontal sinus was explored. The sinus was full of polypoid mucosa and pus, and the opening on the anterior wall was necrosed; the anterior wall was removed and the sinus was packed with bismuth-iodoform-paraffin paste. A week later, at a second operation, right-sided external ethmoidectomy was completed and left-sided fronto-ethmoidectomy was performed through a separate incision. After another two weeks, at a third operation, bilateral radical antrostomy was carried out and the frontal sinus wounds were sutured. By the end of another week the patient's condition was greatly improved; there was neither nasal discharge nor headache, and in a few days she had been discharged to the outpatient department.

Bilateral Otitis Media-Streptococcal Meningitis.

Another patient shown by Dr. Millar was a man who had been admitted to hospital on January 14, 1937, with right subacute otitis media. On paracentesis of the right ear pus was evacuated. On the next day the left ear drum was found to be bulging, and on paracentesis pus was evacuated. There had been a free discharge from the ears on the following day and the patient complained of a frontal headache. Two days later there was also an occipital headache, and the patient was mentally dull; the neck was stiff and the Kernig sign was positive. By lumbar puncture opalescent fluid was obtained under a pressure of over 300 millimetres. Though a smear was "negative", culture resulted in the growth of hemolytic streptococci. In each cubic millimetre of cerebro-spinal fluid 1,500 polymorphonuclear cells and 1,600 lymphocytes were counted. No petrositis could be demonstrated in a skiagram, though the mastoid cells were described as diploietic. On January 21, 1937, Dr. Millar carried out Neumann's operation for meningitis on the right side with a wide exposure of the *dura mater* of the middle and posterior fossae by removal of the petrosal angle and extension towards the petrous apex outside the semi-circular canals. On the left side he had done an incomplete Schwartz operation, draining the antrum. The right ear was regarded as the probable focus. By cisternal puncture twelve cubic centimetres of turbid fluid were removed, and thereafter cisternal puncture was carried out twice a day until January 30 and once a day until February 11. On two occasions during this time lumbar puncture was "dry". On January 25, 1937, the oculist reported the presence of one diopter of papilloedema in each eye, but three days later this had decreased to one-half diopter. On several occasions between January 19 and February 4 the cultures of the cerebro-spinal fluid were positive, but since then all cultures had been "negative".

NOMINATIONS AND ELECTIONS.

THE undermentioned has applied for election as a member of the New South Wales Branch of the British Medical Association:

Wheatley, Arthur, M.B., 1932 (Univ. Sydney), Warialda.

The undermentioned has applied for election as a member of the South Australian Branch of the British Medical Association:

Mackay, Margaret Eleanor, M.B., B.S., 1936 (Univ. Melbourne), Adelaide Hospital, Adelaide.

The undermentioned has been elected a member of the South Australian Branch of the British Medical Association:

Seymour, Stanley Liddell, M.D., C.M., 1935 (McGill), 7, Pembroke Street, College Park.

Public Health.

BOVINE TUBERCULOSIS.

THE following statement is published at the request of the Council of the Veterinary Association of New South Wales.

We, the members of the Australian Veterinary Association, now assembled in conference in Sydney, representing as we do the qualified veterinarians of Australia, consider it necessary, in view of the publicity which has been given to certain views regarding bovine tuberculosis, to issue the following statement:

1. Tuberculosis is one of the three most serious diseases of Australian cattle.

2. It is of importance both from the economic and human health aspects.

3. As to its economic importance, we accept as a reasonable but, if anything, conservative estimate the recent statement of the Director of Veterinary Hygiene of the Commonwealth that the disease costs Australia at present half a million pounds per annum.

4. As affecting human health we accept the evidence of the medical profession that bovine tuberculosis constitutes a grave risk to child life and efficiency.

5. We have shown by actual example that bovine tuberculosis can be eradicated from and kept out of herds and areas, and we know of no reason why such a procedure should not be gradually extended in what would be ultimately, though not for some years, a continent-wide campaign, and we have the example of other countries to support us.

6. We possess in tuberculin a remarkably reliable agent for the detection of tuberculosis when placed in the hands of trained men. Judging from the attitude of the medical and veterinary professions generally towards tuberculin and the published evidence on both sides, the value of the test and the practical consequences which may follow the use of the test are far greater in veterinary medicine than in human medicine.

7. We are satisfied that economically it would be a sound policy gradually to extend an eradication campaign and that for obvious national and humanitarian reasons we consider every effort should be made to conserve our child life against bovine tuberculosis.

8. We are satisfied that no further appreciable improvement in the eradication of bovine tuberculosis can be brought about without the wide and controlled use of the tuberculin test, with appropriate disposal of all affected animals.

9. Whilst recognizing that under existing circumstances it may be necessary to heat milk, we are satisfied that the ideal milk for human consumption is raw milk derived from healthy cows, handled by healthy people, and cooled down to a low temperature as rapidly as possible after withdrawal from the cow, and maintained at a low temperature until delivered to the consumer, and we can see no insuperable obstacle to the ultimate achievement of that ideal.

10. We are satisfied that the surest way to increase the consumption of milk in the cities, a most desirable objective, would be the official assurance that the herds supplying such milk had been subjected to the tuberculin test.

11. We are satisfied that the testing of milk samples for tuberculosis, whilst useful as an indicator in the early stages of a campaign, is not in any way comparable with the tuberculin test as a means of controlling tuberculosis in cattle and safeguarding human health.

12. We would draw attention to the fact that the campaign against bovine tuberculosis is progressing all over the world, and that Australian dairy products, if they are to hold their position, must be the product of healthy cattle.

Obituary.

ROGER ST. CLAIR STEUART.

We are indebted to Dr. A. J. Trinca for the following account of the career of the late Dr. Roger St. Clair Steuart.

It is granted to few members of the medical profession to be the subject of such universal affection and respect as Roger St. Clair Steuart. His death on June 13 after an illness of only a few hours came as a sad blow to all who had been privileged to know him. A serious illness in 1932 and 1933 left him seriously impaired in health, and in the last few months that he spent at his work as in-patient surgeon at the Alfred Hospital it was obvious to all that he was carrying on under difficulties and overtaxing his small reserve of vitality. Finally, in May, 1935, he resigned from the surgical work to which he was devoted, and accepted the position of Senior Medical Officer to the Victorian Railways. Here his health improved considerably, and he made a remark to one of his colleagues only a few days before his death that he had not felt so well for years.

St. Clair Steuart was born in New Zealand in 1882, and after being educated at Wanganui College he graduated at Edinburgh, where he had a distinguished athletic career, being captain of Edinburgh University rugby team and a champion hurdler.

He had a distinguished war record from May, 1915, to the end of the War, and was temporarily attached to the British Expeditionary Force at Suvla Bay. He was twice mentioned in dispatches.

Mr. Steuart's surgery was characterized by absolute honesty and sound judgement, and he spared no pains to help the younger man who had surgical ambitions, and to teach him to shoulder the responsibilities he would have to accept alone in later life.

His public hospital patients received the same unflinching courtesy as his private patients, and admired him as a man. To strike a more personal note, Dick Steuart was a man much beloved by those who knew him well. He had a charm of manner characterized by a disarming smile and keen sense of humour. A sensitive disposition had bred in him a modesty of demeanour which is a bar to progress in this present-day world of self-advancement. Many of us felt that this modesty prevented him reaching the position in his profession that his professional knowledge and skill warranted. There is more in life, however, than financial success, and Dick Steuart has left behind him a memory of a man of gracious manner and courtly bearing, of a generous spirit and scrupulous honesty in his dealings

with his fellow men. He was a perfect gentleman. Our sympathy is extended to his widow and family.

Dr. John Gray writes:

I met Major Steuart for the first time at the Third Australian General Hospital, Abbeville, France. I can see him in the mess, which was in a Nissen hut, walking over to me, a new arrival, and welcoming me in his own genial way and in the traditional manner. He had that fine faculty of making one feel at ease.

"Dick", as we called him, was a great favourite with us all; he had a keen sense of humour and was full of vitality and charm. In the mess, the operating theatre, in the wards, radiating cheerfulness, keen on his work, he gave the benefit of his skill and sympathetic attention to the "diggers" with his kindly smile, chip and jest. His excellent surgery there soon was recognized, and later he

went up to various casualty clearing stations with Major Bryden as anaesthetist, as a surgical team. He made many friends there among the Royal Army Medical Corps surgeons, particularly at the Twenty-First Casualty Clearing Station when his friend Major Anderson, of Dundee, Scotland, was senior surgeon.

I well remember going up to the Twenty-First, which was stationed near Albert, with Major Matthews, who was to relieve him, and the supper party that was given and the affectionate farewell to a man who had won their affection and esteem. Then came the drive back through the snow, when the true character of the individual comes out in friendly discussion of things in general and thoughts in particular. His theatre nursing staff and orderlies would do anything for a man they liked so well.

Major Steuart had a fine physique and had played "rugger" for Scotland in his Edinburgh University days, and he was the best half-back the hospital hockey team ever had.

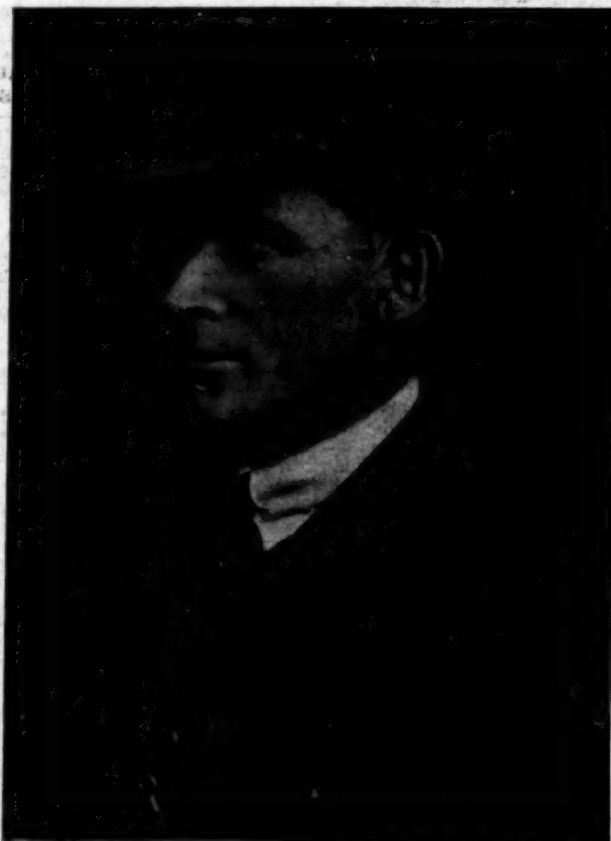
So has passed a man of many parts—an excellent surgeon, a great

sport, a great friend; and we can say in our hearts: "Well played, Dick!"

Dr. G. Raleigh Weigall writes:

The passing of R. St. Clair Steuart has caused a sense of personal loss to all of those who knew him and had worked with him, because to all he was a friend and an example that one liked to have before one. His loss was keenly felt some years ago, when a breakdown in his health forced him to relinquish his practice and his appointment at the Alfred Hospital, to assume less arduous duties than those of a practising surgeon and honorary at a large teaching hospital.

These very duties no doubt were largely responsible for his death at such an early age. He had always given the best of himself to his work, and often, when obviously



not well enough to do so, insisted on carrying his share of the burden of the day and night work associated with his position.

He had gained the highest reputation as an athlete and a sportsman, and later at the War, before the present writer, acting as his house surgeon at the Alfred Hospital, met him first. One was at once struck by the bigness of the man, the bigness of his interests, of his ability, and of his heart.

It was possibly due to the years that he had spent in general practice that he had the more human viewpoint, the more practically helpful understanding of his patients than one whose training had been more purely academic. His ready good-natured humour and his direct and friendly manner to all with whom he worked assured him of the best cooperation, as well as the warmest regard, from patients, nurses, students, residents and colleagues. His house surgeons in particular owed him a debt for the hours of his time that he would spend in "assisting" them at operations, and thus in teaching them operative surgery in a way in which they could not otherwise have learned it. Night after night he would thus spend hours that he could have saved by doing the operation quickly and easily himself, or by leaving it to the unaided efforts of the junior man.

Mr. Steuart had such a sympathetic and understanding nature that the sufferings—surgical or otherwise—of his patients caused him such concern that he might, in a sense, have been said to be lacking in "surgical temperament". But this power he had of considering a patient's domestic, business, financial and other problems along with the surgical ones enabled him in many cases to assess more accurately the importance of the latter, and to arrive at a satisfactory way of dealing with the case as a whole. He thus earned the deepest thanks and lasting friendship of many patients, who were not, to him, just a case of this or that surgical condition, but a fellow being with a lot of troubles, which he shared willingly and dealt with cheerfully and efficiently.

With others who were contemporaries, the writer feels that to have worked with a man like St. Clair Steuart at the beginning of one's professional life was an inspiration and a privilege; to have his help and guidance in some of the problems of practice a little later was of the greatest assistance and encouragement; and to have his example and memory to look back upon will always be valued by those of us who were associated with him at that important and impressionable stage of our careers.

Correspondence.

PROTEIN SHOCK IN GONORRHOEAL OPHTHALMIA.

SIR: Plainly the only justification for inflicting protein shock in *ophthalmia neonatorum* would be an improvement in the visual results. Dr. Waddy was asked for the comparative figures of his or other people's results, with and without the sterilized milk injections, and preferably in Australian conditions. He has not given any at all. With a curious lack of logic he has indeed twice mentioned the numbers of gonorrhoeal ophthalmia in Egypt (which are totally irrelevant), while not giving the significant figures showing the comparative merits of different treatments.

The unavoidable conclusion from this is that he does not know what results he gets or, alternatively, that the results do not support his claims.

Because I do not accept his *ipse dixit* he considers that I have a non-protein shock complex. I must frankly admit to a complex which requires a logical basis of carefully examined fact and which prohibits the blind acceptance of mere opinions, even when proclaimed by Dr. Waddy.

He says it is idle to question Sandford Gifford's opinion. Might I suggest that he study the discussion on *ophthalmia neonatorum* by the British Ophthalmological Society in

1936. Of 18 speakers, many of world-wide reputation, only one made a passing reference to Dr. Waddy's sheet anchor of treatment, and no one advocated it. At least I am in extraordinarily good company.

But what we want is not opinions, we want results. It is common knowledge that excellent results can be obtained with simple lavage and good nursing. Even my humble experience shows twelve consecutive cases with unharmed corneae.

I would suggest to Dr. Waddy that he test for himself the merits of this simpler and more rational treatment before giving any more unnecessary milk injections to new-born babies, and that he publish the results.

Yours, etc.,

Brisbane,
July 13, 1937.

E. O. MARKS.

Congress Notes.

AUSTRALASIAN MEDICAL CONGRESS (BRITISH MEDICAL ASSOCIATION).

Railway Facilities.

It will greatly assist the Railways Department in Melbourne if members travelling by train to attend the Adelaide congress advise the department when they intend travelling from Melbourne, and the accommodation required. The Victorian Railways Department would like to have the following information not later than Saturday, July 31: the number of passengers going to Adelaide from Melbourne, the date of the journey from Melbourne to Adelaide and the accommodation required, whether ordinary first class accommodation without sleeping berth or first class accommodation with sleeping berth. If a Pullman sleeper is desired (the cost being five shillings extra each way), this should be expressly stated.

Queensland members should supply this information to the Victorian Government Tourist Bureau at 204, Adelaide Street, Brisbane (telephone B 6364).

New South Wales members should supply the information to the Victorian Government Tourist Bureau, 11, Martin Place, Sydney (telephone B 1458).

Tasmanian and Victorian members should supply the information to the Secretary of the British Medical Association (Victorian Branch) at 426, Albert Street, East Melbourne.

Members are urged to comply with this request, not only as a help to the Railways Department, but also because they themselves will benefit.

THE ISOBEL BROWN MEMORIAL.

In memory of Dr. Isobel Brown, and at the suggestion of several of her friends, a committee has been formed to establish a permanent memorial to her in connexion with the Rachel Forster Hospital. It is hoped that this will take the form of a room for sick nurses or resident medical officers.

Subscriptions amounting to £128 have already been received from the following: Dr. L. Amphlett, Dr. P. Anderson, Dr. E. Anderson, Dr. A. Aitken, Dr. L. Bevan, Dr. M. Bertram, Dr. J. Brodziak, Dr. M. Cronin, Dr. F. Chenhall, Dame Constance D'Arcy, Dr. L. Dods, Dr. Dillon-Smith, Dr. E. Day, Dr. E. Durie, Dr. Edelsten-Pope, Dr. N. Farrar, Mrs. E. A. Forster, Miss T. Gibson, Dr. G. Gelkie, Dr. N. Gors, Dr. Hoskisson, Dr. M. Hamilton, Dr. E. Leonard, Dr. M. Little, Mrs. Malcolm, Dr. M. McIlraith, Dr. R. Millard, Dr. McClemens, Mrs. McElhone, Dr. W. Nelson, Dr. L. Nicol, Dr. F. Reading, Mrs. Stafford, Dr. T. Saunders, Dr. E. Sandford-Morgan, Dr. Scott-Young, Mrs. E. A. Thomas, Dr. F. Voss, Dr. K. Winning, Dr. Welsh, Dr. A. Walker, Dr. N. Wing.

Dr. E. M. Day, of the Royal Prince Alfred Hospital, is acting as honorary treasurer.

Books Received.

SO I'M A DIABETIC, by Madeleine Scott, A.T.N.A., with a foreword by K. Maddox, M.D., M.R.C.P.: 1937. Australia: Angus and Robertson Limited. Crown 8vo, pp. 67, with illustrations. Price: 3s. 6d. net.

Diary for the Month.

JULY 27.—New South Wales Branch, B.M.A.: Medical Politics Committee.
 JULY 28.—Victorian Branch, B.M.A.: Council.
 JULY 29.—South Australian Branch, B.M.A.: Branch.
 JULY 29.—New South Wales Branch, B.M.A.: Branch.
 AUG. 3.—New South Wales Branch, B.M.A.: Organisation and Science Committee.
 AUG. 3.—Tasmanian Branch, B.M.A.: Council.
 AUG. 4.—Victorian Branch, B.M.A.: Branch.
 AUG. 4.—Western Australian Branch, B.M.A.: Council.
 AUG. 5.—South Australian Branch, B.M.A.: Council.
 AUG. 6.—Queensland Branch, B.M.A.: Branch.
 AUG. 10.—New South Wales Branch, B.M.A.: Executive and Finance Committee.
 AUG. 10.—Tasmanian Branch, B.M.A.: Branch.
 AUG. 13.—Queensland Branch, B.M.A.: Council.
 AUG. 17.—Tasmanian Branch, B.M.A.: Council.
 AUG. 17.—New South Wales Branch, B.M.A.: Ethics Committee.
 AUG. 18.—Western Australian Branch, B.M.A.: Branch.
 AUG. 19.—New South Wales Branch, B.M.A.: Clinical meeting.
 AUG. 24.—New South Wales Branch, B.M.A.: Medical Politics Committee.

Medical Appointments.

Dr. A. M. Rygate has been appointed Government Medical Officer at Woodstock, New South Wales.

Dr. K. C. Donovan has been appointed, pursuant to the provisions of the *Navigation Act*, 1912-1935, Medical Inspector of Seamen at Gladstone, Queensland.

Dr. D. A. J. Hunwick has been appointed Medical Inspector of Seamen, for the purposes of the *Navigation Act*, 1912-1935, at Port Lincoln, South Australia.

Dr. F. W. Fraser has been appointed Medical Officer in the Office of the Director-General of Public Health of New South Wales.

Dr. J. Coffey has been appointed, in pursuance of the provisions of *The Medical and Other Acts Amendment Act* of 1933, of Queensland, Chairman of the Nurses and Masseurs Registration Board.

Dr. R. H. Gundelach has been appointed Medical Officer to the State Government Insurance Office, Brisbane, under the provisions of *The Public Service Act*, 1922 to 1924, *The Insurance Acts*, 1916 to 1934, and *The Workers' Compensation Acts*, 1926 to 1936, of Queensland.

Medical Appointments Vacant, etc.

For announcements of medical appointments vacant, assistants, locum tenentes sought, etc., see "Advertiser", pages xviii to xx.

ADELAIDE CHILDREN'S HOSPITAL, ADELAIDE, SOUTH AUSTRALIA: Resident Medical Officers.
 INNISFAIR HOSPITALS BOARD, INNISFAIR, QUEENSLAND: Assistant Medical Officer.

INSTITUTE OF MEDICAL SCIENCE, ADELAIDE, SOUTH AUSTRALIA: "Neale" Research Pathologist.

MOOROPNA HOSPITAL, MOOROPNA, VICTORIA: Junior Resident Medical Officer.

ROYAL NORTH SHORE HOSPITAL OF SYDNEY, NEW SOUTH WALES: Honorary Physicians.

Medical Appointments: Important Notice.

MEDICAL PRACTITIONERS are requested not to apply for any appointment referred to in the following table without having first communicated with the Honorary Secretary of the Branch named in the first column, or with the Medical Secretary of the British Medical Association, Tavistock Square, London, W.C.1.

BRANCHES.	APPOINTMENTS.
NEW SOUTH WALES: Honorary Secretary, 135, Macquarie Street, Sydney.	Australian Natives' Association. Ashfield and District United Friendly Societies' Dispensary. Balmain United Friendly Societies' Dispensary. Leichhardt and Petersham United Friendly Societies' Dispensary. Manchester Unity Medical and Dispensing Institute, Oxford Street, Sydney. North Sydney Friendly Societies' Dispensary Limited. People's Prudential Assurance Company Limited. Phoenix Mutual Provident Society.
VICTORIAN: Honorary Secretary, Medical Society Hall, East Melbourne.	All Institutes or Medical Dispensaries. Australian Prudential Association, Proprietary, Limited. Mutual National Provident Club. National Provident Association. Hospital or other appointments outside Victoria.
QUEENSLAND: Honorary Secretary, B.M.A. House, 225, Wickham Terrace, Brisbane, B.17.	Brisbane Associate Friendly Societies' Medical Institute. Proserpine District Hospital. Members accepting LODGE appointments and those desiring to accept appointments to any COUNTRY Hospital are advised, in their own interests, to submit a copy of their Agreement to the Council before signing.
SOUTH AUSTRALIAN: Secretary, 173, North Terrace, Adelaide.	All Lodge appointments in South Australia. All contract Practice Appointments in South Australia.
WESTERN AUSTRALIAN: Honorary Secretary, 205, Saint George's Terrace, Perth.	All Contract Practice Appointments in Western Australia.

Editorial Notices.

MANUSCRIPTS forwarded to the office of this journal cannot under any circumstances be returned. Original articles forwarded for publication are understood to be offered to THE MEDICAL JOURNAL OF AUSTRALIA alone, unless the contrary be stated.

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